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An EEG Source-Space Analysis of the Neural Correlates Underlying Self-Regulation

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An EEG Source-Space Analysis of the Neural Correlates Underlying Self-Regulation

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ABSTRACT

Self-regulation is the cognitive process of controlling our thoughts and behaviors to be aligned with our goals. This process is used in many different contexts and has been associated with contributions from several brain regions. This research aimed to investigate differences in four prefrontal areas of the brain while participants applied four different self-regulation strategies. We recorded EEG while participants (N = 132) performed three tasks which engaged each of the four self-regulation strategies: the AX-CPT task engaged proactive and reactive control, the Go/Nogo task engaged inhibitory control, and the hybrid Flanker Global/Local task engaged the resolution of response conflict. This study used the N2 event-related potential (ERP) to capture the neural activity related to each self-regulation strategy and then source-space analyses (eLORETA) were conducted to estimate the activity in four regions of interest (ROIs): dorsolateral (DL) PFC, ventrolateral (VL) PFC, ventromedial (VM) PFC, and dorsal ACC. The dorsal ACC was most activated for proactive control, indicative of performance monitoring. The right VLPFC was indicative of conflict adaptation in reactive control and response conflict, and indicative of motor inhibition in inhibitory control. DLPFC was most active for goal maintenance during proactive and reactive control. The left VLPFC was most active during reactive control, indicating its importance in memory of goal information. These results are in line with much of the previous literature. VMPFC did not show any differences across the strategies likely due to the lack of emotional context. This study builds on the extant literature by directly comparing neural processes across four different self-regulation strategies within one large sample, highlighting the fact that various self-regulation strategies recruit unique patterns of activation and thus future research should not collapse across these strategies.

Keywords: *self-regulation, ERPs, source-space analysis, cognitive neuroscience*

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CHAPTER 1

INTRODUCTION

Self-regulation is a cognitive process used to maintain and achieve goals, to this end, it is the mechanism by which a person controls their thoughts and behaviors to be context appropriate (for a review see Nigg, 2017). More broadly, self-regulation encompasses many executive processes, such as attention, inhibition, emotion-regulation, etc. and can be broken down into several cognitive strategies used to achieve a goal state. Four strategies that were the focus of this study were proactive and reactive control, inhibitory control, and response conflict. Braver (2012) outlined the differences between proactive control (PC), which is the maintaining of self-regulation prior to an anticipated outcome, and reactive control (RC), which is the activation of self-regulation following the detection of an outcome. Proactive control is used to enhance performance in strategic action strategies, while reactive control is used to enhance performance in short-term adjustments to changes in the environment. These two strategies have typically been studied in the context of the Expectancy "AX" continuous performance task (AX-CPT; MacDonald & Carter, 2003), where participants engage in both strategies in different trials (the full description of the task is detailed below in the Methods section). Inhibitory control (IC) is the ability to suppress irrelevant information and behaviors in the context of a particular goal. This strategy has been studied extensively in the context of the Go/Nogo task (Garavan et al., 1999), where participants either produce a response or inhibit their response to a stimulus. The strategy to resolve response conflict (RespC) is engaged when there is a competition for two responses, a correct response and one that has to be overridden (Botvinick, Cohen, & Carter, 2004). This strategy can be studied in several contexts, but the one that was used in this study was in the context of a hybrid flanker (Eriksen & Eriksen, 1972) and global/local task (Navon,

1977). In this particular task there are trials with incongruent stimuli where participants are conflicted with two equally probable potential responses. All of these strategies are examples of the brain receiving information from the environment and updating behavioral responses through the mechanism of self-regulation. However, the neural mechanisms by which these strategies differ in individuals is largely unknown. It is critical to investigate how the brain engages in self-regulation, particularly with respect to the differences in brain networks involved in different contexts of self-regulation. To that end, research into these mechanisms aims to advance our understanding of the differences in the brains of those affected by mental health disorders.

Self-Regulation Failure and Mental Health

Self-regulation has been widely studied across the fields of social, personality, developmental, and cognitive psychology as well as neuroscience and clinical science. The breakdown of self-regulation plays a critical role in many psychological disorders, such as attention deficit/hyperactive disorder (ADHD; Shiels & Hawk Jr., 2010), bipolar disorder (Tseng et al., 2015), depression (as reviewed by: Paulus, 2015; Wang, Chassin, Eisenberg, & Spinrad, 2015), schizophrenia (Boudewyn & Carter, 2018; Orellana & Slachevsky, 2013), autism spectrum disorder (Bachevalier & Loveland, 2006), obsessive-compulsive disorder (Fineberg et al., 2014), substance-use disorder (Fleming & Bartholow, 2014; Zucker, Heitzeg, & Nigg, 2011), among others. Therefore, research into the mechanisms of self-regulation, specifically the neural mechanisms, is essential to understanding the biological sources underlying these deficits in mental health.

ADHD has been studied in-depth as an example of self-regulation deficits resulting in behavioral control problems. This disorder is commonly associated with a failure of self-

monitoring in order to adapt behaviors to be context appropriate (Shiels & Hawk Jr., 2010). Neuroscience studies have investigated differences in anterior cingulate cortex (ACC) activity in those with ADHD using the error-related negativity (ERN), an event-related potential (ERP) that has been theorized to be generated by the ACC (Dehaene, Posner, & Tucker, 1994) and is thought to reflect error-processing, with greater negative amplitudes appearing after an error has been exhibited in a task (Holroyd & Coles, 2002). Several studies have shown reduced ERN amplitudes in ADHD participants compared to controls (Albrecht et al., 2008; Liotti, Pliszka, Perez, Kothmann, & Woldorff, 2005; Van De Voorde et al., 2010). These changes are generally in areas of the brain involved in self-regulation, such as the ACC, suggest that mechanisms of self-regulation are altered in those with ADHD.

Another example of a disorder related to self-regulation is addiction (Bechara, 2005; Koob & LeMoal, 2008; Sayette, 2004). Those with a drug addiction struggle to override drug use behaviors in order to achieve a more positive outcome, such as quitting smoking. This failure of self-regulation manifests in several ways, such as the inability to appropriately monitor behavior to prevent relapse, inability to exert self-control over smoking behavior, and the misuse of smoking behavior to address other problems in the user's life (Sayette, 2004). The neuroscience research into differences in self-regulation related to smoking have been mainly in the prefrontal cortex (PFC) regions of the brain. These areas of the brain, such as lateral PFC and ACC, are recruited when self-regulation is required, for example, when smokers have to inhibit their cravings (Brody et al., 2007). Many theories suggest that the failure of top-down regulation of the reward areas of the brain by PFC regions is what results in a failure of self-control by those with an addiction (Bechara, 2005; Demos et al., 2011; Koob & Le Moal, 2008). As a whole, addiction and ADHD are both important examples of atypical behaviors resulting from flawed

self-regulatory mechanisms. More research into the differences in self-regulation strategies, specifically brain activation differences, could help create more targeted research studies on self-regulatory brain areas in the context of these mental health disorders.

Brain Regions Involved in Self-Regulation

Scientists have been researching the involvement of the PFC regions in the brain as the center-stage for self-regulation since the famous case of Phineas Gage in the 1800s (as reviewed by Heatherton, 2011). Although his was not a localized case, Gage's frontal lobe was severely damaged when a railroad spike went through the front of his head. He survived the accident but went through a profound personality change, where he was previously seen as nice and hardworking, after the accident he was easily angered and violent. The damage to this area of the brain resulted in behavioral changes that showed a distinct lack of self-regulation. More recently, many functional magnetic resonance imaging (fMRI) studies, as well as electroencephalography (EEG) studies using source-space analysis (inverse model of cortical activation based on patterns of scalp EEG), have examined the relationship between self-regulation and prefrontal brain areas, particularly the lateral PFC (VLPFC, ventrolateral PFC; DLPFC, dorsolateral PFC), ventromedial PFC (VMPFC), and the ACC (for review see Banfield et al., 2004; Krendl & Heatherton, 2009).

The lateral PFC has several subareas but there are two main areas of interest for self-regulation: DLPFC and VLPFC. DLPFC has been theorized to be involved with attentional processes activated when engaging in the attentional demands of a task (D'Esposito & Postle, 1999; MacDonald, Cohen, Stenger & Carter, 2000). MacDonald and colleagues (2000) found that (left) DLPFC was active in maintaining task information during a Stroop task to maintain a

representation of the context and engage strategic control. Their results showed DLPFC to be implementing top-down control of behaviors required for a particular task. Braver, Paxton, Locke, and Barch (2009) showed differential activation patterns for left DLPFC during proactive and reactive control and suggested that these activations were flexible within individuals in exhibiting self-regulation. It has also been theorized that DLPFC's role in attention and top-down control is involved in updating goal information (Miller & Cohen, 2001; Sakai & Passingham, 2006). The DLPFC functions can be examined separately for the left and right hemispheres as well. Vanderhasselt, De Raedt, and Baeken (2009) examined several studies where DLPFC lateralization was observed during the Stroop task and discussed that in several studies the results indicated that the left DLPFC was involved in attentional preparation of conflict during a task (MacDonald et al. 2000; Aarts, Roelofs, & van Turennout, 2008). In these studies (using a task-switching Stroop task) the left DLPFC activity was not related to the amount of conflict during the trial (congruent or incongruent) but instead related to preparation of potential conflict during the cue. This is in line with the previous studies showing this area to be maintain task and goal information. The right DLPFC was also discussed in Vanderhasselt et al. (2009) and examined across several studies. Specifically, in Kerns et al. (2004), the right DLPFC was more active during trials that had fast reaction times after incongruent trials or error trials, furthermore there was a positive correlation between this activation and accuracy. Therefore, the right DLPFC was involved when there was more attentional conflict.

The VLPFC area of the brain has been studied both in the left and right hemisphere and shown to have differential functions in each hemisphere of the brain. When studied in the context of self-regulation, the left VLPFC has been shown to contribute to the control of memory and keeping goals and actions in memory during task execution (Badre et al., 2005, Badre &

Wagner, 2007). In line with this theory, Braver and colleagues (2007) theorized greater left VLPFC activation during trials where reactive control was engaged based on the findings of D'Esposito and colleagues (1999). During reactive control, it is critical to hold the previous stimulus in memory to change or update the response to achieve the goal outcome. In this way, the left VLPFC allows for flexible self-regulation to maintain a goal. The right VLPFC also contributes to self-regulation by engaging during motor inhibition (Aron, Robbins & Poldrack, 2004) and reflexive reorienting (Corbetta, Patel, & Shulman, 2008). In an MRI meta-analysis by Levy and Wagner (2011), they examined studies that used Go/Nogo and Stop Signal tasks to study motor inhibition, and the Posner Cueing and Oddball tasks for reflexive reorienting. They indeed found more right VLPFC activation during both motor inhibition tasks and reflexive reorienting tasks and found the patterns between them to be similar. Therefore, the right VLPFC seems to contribute to self-regulation strategies engaged where there are abrupt perceptual changes during a task, such as those engaged to stop or override motor responses or when attentional control is needed for stimuli outside of the focal area. Additionally, right VLPFC appears to be engaged during response conflict and conflict adaptation, shown during a set of Stroop task experiments by Egner (2011). This study also indicated that the right VLPFC activity was supplemented by DLPFC activity. Thus, the interaction between these two brain areas may contribute to differences in individual self-regulation strategies.

Another area of the brain that contributes to self-regulation and has been shown to be involved in the self-control network with the DLPFC is the VMPFC (also known as the medial part of the orbitalfrontal cortex, OFC). Broadly, the VMPFC is thought to be important in emotional and behavioral regulation (Dolan & Park, 2002). Within behavioral regulation, VMPFC has been shown to be involved during goal-directed decisions (Hare et al., 2008; Rolls,

McCabe & Redoute, 2008). Along this line, Hare, Camerer, and Rangel (2009) showed that the VMPFC is modulated by DLPFC and differences in this modulation result in differences in self-control. In their study, dieters engaged in decision-making requiring self-control, while fMRI data was collected. They theorized that the VMPFC takes factors from the environment and values them in the context of the goal, and that the DLPFC modulates these values in a long-term context. Those with damage to the VMPFC show a lack of control over emotional expression (Stone, Baron-Cohen & Knight, 1998) and disregard for others (Blair and Cipolotti, 2000). Taken together, these studies suggest that the VMPFC is involved in self-control related to decision-making that results in behavioral control.

The final brain area of interest for this study of self-regulation mechanisms is the dorsal ACC. The ACC has been thoroughly studied in the context of conflict monitoring (Gehring & Knight, 2000; MacDonald et al., 2000) and error processing (Carter et al., 1998; Menon et al., 2001). Generally, ACC activity is related to updating behaviors in response to conflicts and integrating errors into future behavior. Botvinick, Cohen, and Carter (2004) reviewed ACC activity and discussed that the ACC is associated with response conflict, specifically during a Stroop task on incongruent trials (where competition between two responses is high). However, ACC activity has also shown to be related to incongruent trials during the standard Global/Local task (Weissman et al., 2003) and a hybrid flanker Global/Local task (Lux et al., 2004; their described task has similarities to the task in the current study but required different responses and had an attentional aspect). Furthermore, the ACC has been studied in the context of those low in self-regulatory control, such as those with ADHD. Bush et al. (1999) showed lower ACC activity for those with ADHD than controls during a counting Stroop task. Therefore, inefficient processing in this area of the brain may be related to differences in self-regulation processing.

It is clear that all four of these prefrontal brain areas are involved in several aspects of self-regulation. Many of the above-mentioned studies used different tasks to examine self-regulation in different contexts; however, none of them examined these contexts in a single sample. Current research suggests that different self-regulation strategies rely on a similar set of neural mechanisms (Gratton et al., 2018); however, there is little research focusing on comparing brain activation differences across different self-regulation strategies. One key article that was used to develop this study was by Wagner et al. (2005), where the authors were also interested in testing whether similar or distinct brain activity was found across different self-regulatory tasks. They found several common brain areas that were activated across three self-regulatory tasks and specific areas were recruited to reflect any differences in performance. However, their study focused solely on response inhibition as their self-regulatory strategy, so they were looking at what was common to response inhibition across three tasks. They also only had a sample of 14 participants and since they used fMRI, they were not able to investigate the precise timing of self-regulation. What the literature is lacking is an analysis of brain activations engaged during several different self-regulation strategies within individuals. Many of the studies of prefrontal brain regions mentioned above used fMRI to investigate them; however, some used EEG to study differences in these regions using source-space analysis techniques. Source modeling uses EEG to *estimate* where in the brain certain ERP activity is originating. In conjunction with the excellent temporal resolution of EEG, these techniques can expand on some of the research done with fMRI like Wagner et al. (2005). In the present study, I used source modeling to estimate what brain areas were involved during the precise moments certain self-regulation strategies were engaged.

The focus in this study was on the sources of brain activation underlying a particular ERP, the N2, a component frequently associated with self-regulation. This component is a negative deflection that usually occurs 200-400 milliseconds post-stimulus (Luck, 2014) at mediofrontal electrodes. The N2 component has been shown to reflect several different self-regulation processes (Lamm, Pine, & Fox, 2013; Nieuwenhuis et al., 2003; Wouwe, Band, & Ridderinkhof, 2009) and has been shown to be associated with changes in all four self-regulatory strategies that were the focus of this study (Falkenstein, 2006; Lamm et al., 2013). To examine the potential changes in brain regions related to these strategies, we collected EEG during three behavioral tasks that specifically recruited each of the four self-regulation strategies: inhibitory control in the Go/Nogo task, proactive control and reactive control in the AX-CPT task, and response conflict in the hybrid Flanker Global/Local task. All tasks are described in more detail in the Methods section.

Hypotheses

Based on the literature reviewed above, I hypothesized that there would be differences in neural activity in the six brain regions of interest (ROIs), based on the differences in participants' engagement in self-regulatory strategies.

1) I expected that DLPFC activity would differ in modulation of reactive and proactive control based on the findings of Braver, Paxton, Locke, & Barch (2009). More specifically, I expected greater left DLPFC activation in proactive control than reactive control and greater right DLPFC activation in reactive control than proactive control (Vanderhasselt, De Raedt & Baeken, 2009).

2) I expected VMPFC activity would be recruited more during inhibitory control (Hare et al., 2008).

3) I expected that left VLPFC would have higher activation during reactive control (Braver et al., 2007), while right VLPFC would be more active during inhibitory control (Aron, Robbins & Poldrack, 2004) and response conflict (Egner, 2011).

4) Finally, I expected dorsal ACC activity to be to be more engaged during response conflict and during reactive control (Lamm, Pine, & Fox, 2013).

CHAPTER 2

METHOD

Participants

287 undergraduate students (120 males, 165 females, and 2 who selected "other") were recruited from the University of Arkansas general psychology course to participate in this study. All students were given course credit for their participation. Ethical approval for the study was obtained from the University of Arkansas' Institutional Review Board (1708026820). The mean age was 19.5 years (*SD* = 2.63, range = 18-47). The majority of the participants were right-handed (89%). The participant demographics included 80% Caucasian/White, 13% Hispanic/Latino, 8% multi-racial, 4.5% Asian, 3% African American/Black, >1% Native American, >1% Middle Eastern/Northern African. Criteria for exclusion from participating in the study were current psychiatric diagnosis, current use of psychoactive medication, uncorrected visual impairments or hair styles that would not allow an electrode to be directly placed on the scalp. These hair styles included but were not limited to extremely thick hair, thick tight braids, dreadlocks, or sewn in hair. We prescreened for these through the University of Arkansas Sona System. After data collection, participants were also excluded from analyses if any of the task conditions contained fewer than 10 correct artifact free trials (anything less created too low of a

signal-to-noise ratio for ERP analyses) or had low accuracy in any of the conditions. For AX-CPT and Go/Nogo tasks, participants with less than 20% accuracy were removed. For the hybrid Flanker Global/Local task, participants with less than 40% accuracy were removed. Each of these cutoffs excluded participants greater than about two standard deviations from the mean accuracy. Finally, an additional participant was excluded as an outlier based on Cook's distance criterion of greater than 4/N (N = number of observations). The final sample analyzed in this study included 132 undergraduate students (59 males, 72 females, and 1 who selected "other"). The sample demographics included 79% Caucasian/White, 10% Hispanic/Latino, 7.5% multi-racial, 6% Asian, 2.25% African American/Black, >1% Native American. No significant group differences were found between the age, gender, or racial/ethnic backgrounds of participants who were included in the final sample compared to those who were excluded from further analysis.

Procedure

The procedure followed what was published by Rawls et al. (2018) and Eisma (2020). Participants were first introduced to the experimental environment and written informed consent was obtained. Participants then completed a battery of questionnaires while seated in the testing room. After the first set of questionnaires, participants were seated 67 cm in front of a computer monitor and the electrode sensor net was applied to their head. They then completed two practice blocks for each of three behavioral tasks to ensure they understood the task procedure. If the participants indicated they still did not understand, then the practice blocks were repeated. These three tasks were broken up into roughly 50-trial blocks that varied pseudo-randomly in presentation order (but all participants received the same trial order) and after each block participants took a break before beginning the next block. After completing all the task blocks, the electrode sensor net was removed, and a second set of questionnaires was completed.

Following this second set, participants completed a Competitive Reaction Time (CRT) Task based on the Taylor Aggression Paradigm (which was not analyzed for this project and therefore will not be discussed) and upon completion filled out a third and final set of questionnaires. The entire study session on average took four hours to complete.

Measures

AX-CPT. The Expectancy "AX" continuous performance task (AX-CPT; MacDonald & Carter, 2003) was used to assess proactive and reactive control. In this task, pairs of letters were presented, one following the other. The first letter (A or B) served as the cue and the second letter (X or Y) served as the probe. There were four trial types within this task, A-X, B-X, A-Y, and B-Y. Participants were told to press the first button on a five-button box as soon as they saw the first letter (the cue) on the screen. Then once they saw the second letter, the probe, they pressed either the first button or the fifth button as fast as they could. If they saw the target letter pair, A-X, they were instructed to press the first button and then the fifth button. But, if they saw any other pair of letters (B-X, A-Y, or B-Y) they were instructed to press the first button and then the first button again. Two practice blocks of 10 trials were presented before the task began. The practice blocks for this task included error feedback (a red dash appeared at fixation) following each trial if participants entered the wrong response or a response was too slow. The task consisted of eight blocks and each block contained 58 trials. Within each block, 70% of the trials contained A-X pairs, with the other three pairs equally appearing on 10% of the trials to make up the other 30% of trials within the block. Order of presentation of these pairs was pseudo-randomized within blocks. Since the A-X pairs were presented the majority of the time, this requires participants to use specific self-regulation strategies to respond appropriately during the other pairs of letters. During the B-X trials, participants had to maintain the memory of the B

cue in order to respond correctly to the X probe, which required proactive control. During A-Y trials, participants were primed with an A cue and had to adapt to the presentation of the Y probe when they expected an X probe, which required reactive control.

All stimuli were presented on a 17" monitor using E-prime Software (Psychology Software Tools, Inc., Pittsburgh, PA; Schneider et al., 2002). Stimuli were shown on a black screen. Each trial started with a fixation screen lasting 500 ms followed by the cue, which was displayed for 400 ms. After the cue, a delay fixation screen was presented for 2000 ms, followed by the probe, which was displayed for 400 ms. A fixation screen was displayed for an inter-trial interval that was jittered between 1000 – 2000 ms while participants waited for the next trial to begin. This timing variation ensures a variation in the phase oscillation upon which a stimulus falls from trial-to-trial to accurately capture event-related potentials. Cue and probe letters were presented in 60-point size uppercase bold Courier New font, with cue letters presented in blue font and probe letters presented in white font to help participants remember the letter order (see Figure 1).

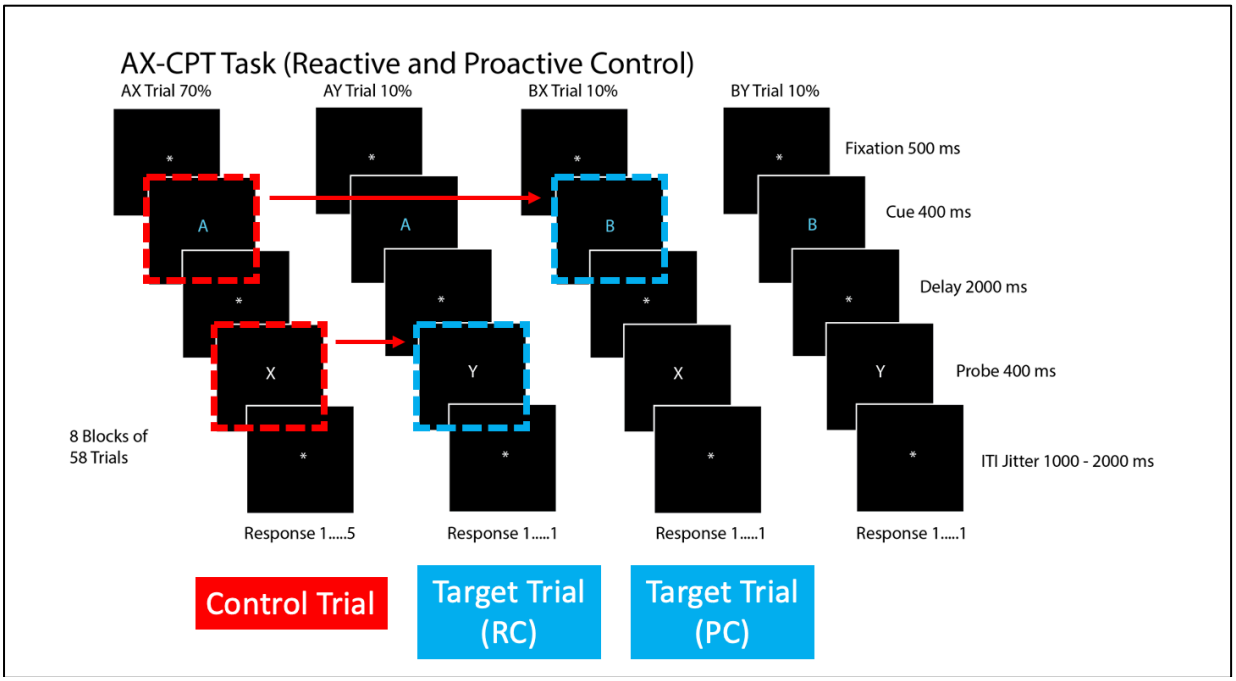

Figure 1. Task diagram of the AX-CPT task. The dashed boxes indicate the time-locked stimuli used for ERP analyses (note: the dashed boxes are for demonstration purposes; they were not shown in the task). The target condition stimuli are shown in blue and the control condition stimuli are shown in red. Here, "B" of B-X trials was the target stimulus for proactive control (PC) and its respective control trial was the "A" of A-X trials. The "Y" of A-Y trials was the target stimulus for reactive control (RC) and its respective control trial was the "X" of A-X trials.

Go/Nogo Task. The Go/Nogo task was used to assess inhibitory control. The task was adapted from one used by Garavan et al. (1999). All stimuli were displayed using E-prime software as described in the section above. On each trial a white letter stimulus was presented at the center of the screen. Participants were told to respond on the button box as quickly and accurately as possible to each letter (Go stimuli) except if the letter that appeared was an "X" (Nogo stimulus), in which case they were told not to respond. Before the task began, participants completed one practice block of 10 trials.

The task consisted of five blocks of 53 trials each, where 75% of trials in each block contained Go stimuli and 25% of trials in each block contained Nogo stimuli. The order of these trial types was pseudo-randomized within blocks. Each trial began with a fixation screen that lasted 100 ms, followed by the stimulus, which displayed for 200 ms. A fixation screen then

appeared for 600 ms, while participants responded and then an inter-trial interval jittered from 0-500 ms (see Figure 2).

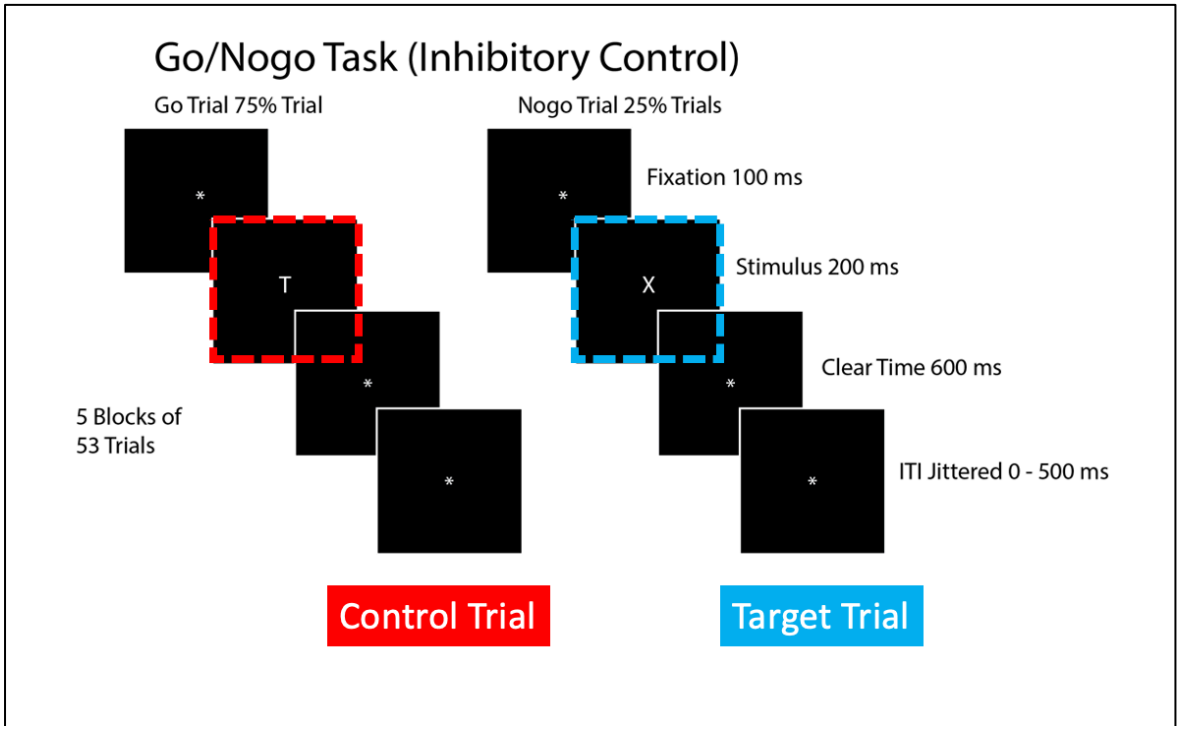

Figure 2. Task diagram of the Go/Nogo task. The dashed boxes indicate the time-locked stimuli used for ERP analyses (note: the dashed boxes are for demonstration purposes; they were not shown in the task). The target condition stimuli are shown in blue and the control condition stimuli are shown in red. Here, "X" of Nogo trials was the target stimulus for inhibitory control and its respective control stimulus was the letter stimulus (e.g. "T") in the go trials.

Hybrid Flanker Global/Local Task. This task was used to assess response conflict and was adapted from Navon (1977) and Eriksen and Eriksen (1972). I used this hybrid task because previous studies from our lab indicated that the canonical Flanker task may not be difficult enough in undergraduate samples to evoke the standard congruent – incongruent difference. During the task instructions, participants were instructed to respond with the first button on the button box if they saw stimuli containing "H"s or the fifth button of the button box if they saw stimuli containing "S"s. At the beginning of every trial, participants were first cued by seeing the word "Big" (global cue) or "Small" (local cue). Following the cue, each trial contained either a congruent stimulus where both the global and local information led to the same response (e.g. a

global H made of local Hs or a global S made of local Ss) or an incongruent stimulus where the global and local information led to a different response (e.g. a global H made of local Ss or a global S made of local Hs). Therefore, on congruent trials, the response was the same regardless of the cue. However, on incongruent trials the cue indicated to which information (global or local) participants were required to respond. Before the task began, participants completed two practice blocks of the task. The only difference in the practice blocks and the actual task was that the congruent and incongruent stimuli were displayed longer in the practice, so that participants could make sure they saw the global and local differences in the stimuli.

This task contained six blocks of 48 trials each. All four trial types (global congruent, local congruent, global incongruent, and local incongruent) were presented equally (25% of trials) within each block. The order of these trial types was pseudo-randomized within blocks. Each trial began with a global/local cue, which was displayed for 2000 ms. Then, the letter stimulus was displayed for 200 ms followed by a fixation screen that appeared for 1100 ms. Finally, an inter-trial interval fixation screen was jittered for 0-500 ms. Participants were instructed to respond as quickly and accurately as possible. All stimuli were presented using E-prime software as described in the AX-CPT section. The letter stimuli were displayed as white capital letters in Arial font (see Figure 3).

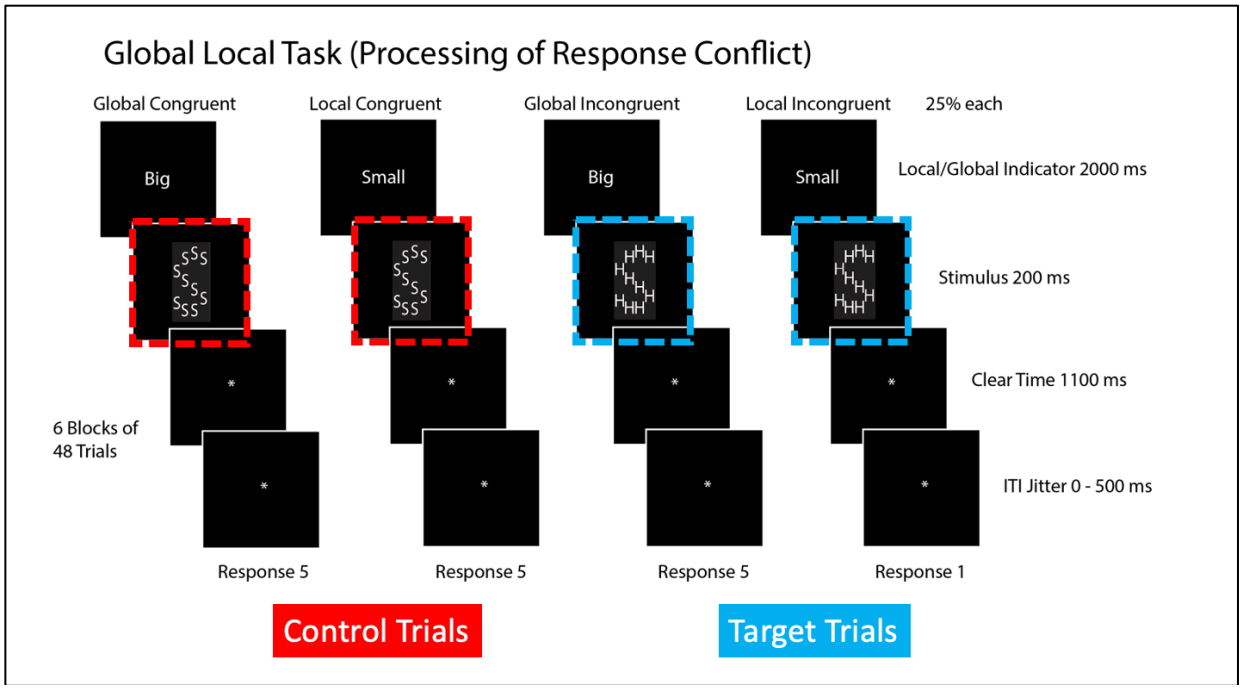

Figure 3. Task diagram of the modified hybrid Flanker Global/Local task. The dashed boxes indicate the time-locked stimuli used for ERP analyses (note: the dashed boxes are for demonstration purposes; they were not shown in the task). The target condition stimuli are shown in blue and the control condition stimuli are shown in red. Here, incongruent trials (global "S" made of local "H"s) were the target stimuli for response conflict and the respective control trials were the congruent trials (both global and local "S"s).

EEG Data Collection and Processing

EEG data collection procedures were consistent with Lamm et al. (2013). EEG data was recorded using a 128-channel Geodesic Sensor Net and sampled at 1000 Hz using EGI software (Net Station; Electrical Geodesic, Inc., Eugene OR). After all channel impedances were reduced to below 50 kΩ, the recording started, and all channels were referenced to Cz.

All EEG data were pre-processed in MATLAB's processing toolbox EEGLAB (Delorme & Makeig, 2004; http://www.sccn.ucsd.edu/eeglab), using a pipeline developed by Dr. Eric Rawls (Eisma, 2020). The data were first downsampled to 125 Hz. Then a zero-phase Hamming windowed-sinc FIR filter was applied to the data as a bandpass filter from 0.1 – 35 Hz. EEG channels were removed and later interpolated if the joint probability of that channel's data and all channel data exceeded four standard deviations from the mean. Then the data were segmented

from -300 – 900 ms and stimulus-locked around each of the target stimuli associated with the four self-regulatory strategies as well as the appropriate control condition stimuli. Proactive control target segments were time-locked to the presentation of the "B" cue of the B-X trials and the control condition segments were time-locked to the presentation of the "A" cue of the A-X trials. Reactive control target segments were time-locked to the presentation of the "Y" probe of the A-Y trials and the control condition segments were time-locked to the presentation of the "X" probe of the A-X trials. Inhibitory control target segments were time-locked to the presentation of the Nogo stimuli and the control condition segments were time-locked to the presentation of the Go stimuli. Response conflict target segments were time-locked to the presentation of the incongruent letter stimuli (averaged across global and local conditions) and the control condition segments were time-locked to the presentation of the congruent letter stimuli (averaged across global and local conditions). Each segment was baseline corrected across the entire segment by mean-centering. Infomax ICA was run on this cleaned dataset using *runica* (Makeig, Jung, Bell, Ghahremani, & Sejnowski, 1997) and using the ADJUST plugin (Mognon et al., 2011) to identify and remove artifactual components containing eye blinks, eye movements, and other stereotyped sources of motion artifacts. The cleaned segments were then examined for any further artifacts (such as fast transits) and were rejected with a threshold of ±140 μV. Finally, all removed channels were interpolated using spherical interpolation and all segments were averaged referenced. The N2 component means were only extracted from correct trials. Grand average waveforms were created for each self-regulatory strategy (averaged across the target conditions and control conditions to decrease selection bias) to select the N2 component time window (with 0 ms indicating stimulus onset; Figure 4). Then scalp distributions were created for all 128 electrodes to ascertain for which electrode the N2

component was maximal (Figure 4). Typically, the N2 is extracted from electrode FCz, so that electrode was chosen to align with the literature.

Figure 4. Grand averaged waveforms for the N2 for each strategy. For visualization purposes, the target condition is shown in red and the control condition is shown in blue. Each N2 time window (shown in gray) was examined at the electrode where it showed maximum amplitude based on the scalp topography (FCz = electrode 6). Here, the frontal negativity is not as concentrated at electrode 6 for three of the strategies because it seems that the posterior P3 (positive) component is washing out the negative amplitudes (making the N2s less negative). The source-space analyses get around this issue.

Statistical Analysis

Behavioral Data Analyses. Accuracies (percentage correct) and reaction times (RT) were examined for each condition, target vs. control, for each of the four self-regulation strategies using paired samples *t*-tests. Because two of the self-regulation strategies, proactive and reactive control, have the same control condition (AX), they were not compared to each other when analyzing accuracy and reaction time. Instead, each was compared to the other strategies using several 2 (target vs. control) x 2 (PC vs IC; PC vs. RespC; RC vs. IC; RC vs. RespC) ANOVAs. For reaction times, the self-regulation strategy, inhibitory control, was left out of analysis because in the Go/Nogo task, participants do not respond on Nogo trials, so there was no target condition.

N2 Analysis. Mean N2 amplitudes were extracted for each of the target and control conditions for each of the four self-regulation strategies. The mean amplitude N2s during the target condition were then analyzed as the dependent variable (DV) of a multilevel regression model conducted in R using *lme4* (Bates, Mächler, Bolker, & Walker, 2015), with a random intercept at the participant level to account for the repeated measures design. Covariates were included for: control condition N2 mean amplitude and accuracy during the target conditions. The control condition N2 means were included as a covariate to remove variance related to N2 activity unrelated to self-regulation and the target accuracy covariate was included to remove variance related to differences in accuracy across self-regulation strategies (accuracy was taken as a proxy of effort; thus, removing variance associated with a specific task being more difficult, i.e. difficulty related changes in brain activation). Finally, an independent variable (IV) with four levels of self-regulation strategy was included in the model to identify strategy differences in N2 mean amplitudes after controlling for the covariates. Post-hoc contrasts were compared across the levels of self-regulation strategy using *emmeans* package in R (Lenth, 2020) with a Bonferroni correction for multiple comparisons.

Source Space Analyses. To analyze source space activation, I followed a pipeline that was used by Buzzell et al. (2017). This included registering standardized electrode locations for a 128 channel Geodesic Sensor Net within MRI-space, segmenting MRI volumes, then constructing a source and head model, calculating a distributed inverse model, constructing regions of interest (ROIs) to analyze current density, and statistically analyzing the current density values within the ROIs. All source space activations were constructed using the Fieldtrip software library (Oostenveld et al., 2011). Since I did not collect individual MRI scans for our participants, an averaged adult MRI template was used from the Montreal Neurological Institute

(MNI) with one-millimeter (mm) resolution (MNI152_T1_1mm.nii). Next, the MRI was segmented using the automated packages in Fieldtrip (*ft_volumesegment*) to extract just the head and brain surfaces from the MRI. Continuing within Fieldtrip, a five mm resolution volumetric source model grid was created within the averaged MRI-space from the segmented gray matter (*ft_prepare_sourcemodel*). A head model was then constructed to properly describe electrical current flow through the head using the finite element method (FEM). A hexahedral mesh was created within the averaged MRI-space and labeled with one of five brain/head areas (gray and white matter, CSF, skull or scalp). A "SIMBIO" FEM head model was then created (*ft_prepare_headmodel*) which assigns conductivity values with the associated tissue type. Then, the electrode locations were fit to the MRI template by identifying four anatomical fiducials, Nz, LPA, RPA, and Cz. Next, the lead field matrix (forward solution) was created using the source model, head model, and electrode locations (*ft_prepare_leadfield*). The inverse model was calculated (*ft_sourceanalysis*) using the exact low-resolution electromagnetic tomography (eLORETA) method (Pascual-Marqui, 2007; Pascual-Marqui et al., 2011). Source-space modeling techniques (specifically the use of sLORETA and eLORETA) have been thoroughly validated computationally (Pascual-Marqui, 2007), through comparisons with simulations (Mikulan et al., 2020), and through their use in identifying epileptic foci for surgery (Michel & He, 2019; Sohrabpour et al., 2015). This created a dipole moment vector for each source volume grid location, which was then converted into a power value and current source density reconstruction (CDR) values ($\mu A/mm^3$) were extracted for each N2 mean amplitude. To ensure that the CDR values (which are current values instead of voltage) were representing negative values of N2, the immediately preceding positive peak before the N2 was selected and subtracted its mean peak from the N2, separately for each self-regulation strategy condition and separately

for each target and control condition. These CDR values were exported for regions of interest (ROIs), generated using the Brainnetome MRI atlas (Fan et al., 2016). I examined six ROIs related to self-regulation: dorsal ACC, DLPFC (right vs. left hemisphere), VLPFC (right vs. left hemisphere), and VMPFC (Table 1, Figure 5). Each ROI was matched to areas used in studies examining these self-regulation strategies and also had corresponding Brodmann areas to those studies (Table 1).

Table 1

Regions of interest for the analysis of CDR data

DLPFC	
(Braver, Paxton, Locke, & Barch, 2009)	
Brainnetome anatomical descriptions:	A9/46d, dorsal area 9/46
Brodmann Area:	9
Left MNI coordinates:	-27, 43, 31
Right MNI coordinates:	30, 37, 36
VLPFC	
(Aron, Robbins & Poldrack, 2004; Badre & Wagner, 2007)	
Brainnetome anatomical descriptions:	A12/47o, orbital area 12/47
Brodmann Area:	47
Left MNI coordinates:	-36, 33, -16
Right MNI coordinates:	40, 39, -14
VMPFC	
(Hare et al., 2008)	
Brainnetome anatomical descriptions:	A11m, medial area 11
Brodmann Area:	11
Left MNI coordinates:	-6, 52, -19
Right MNI coordinates:	6, 57, -16
dACC	
(MacDonald et al, 2000)	
Brainnetome anatomical descriptions:	A24cd, caudodorsal area 24
Brodmann Area:	24
Left MNI coordinates:	-5, 7, 37
Right MNI coordinates:	4, 6, 38

Note. Left and right hemisphere source data (CDR values) were extracted separately for DLPFC and VLPFC and collapsed across hemispheres for VMPFC and dACC.

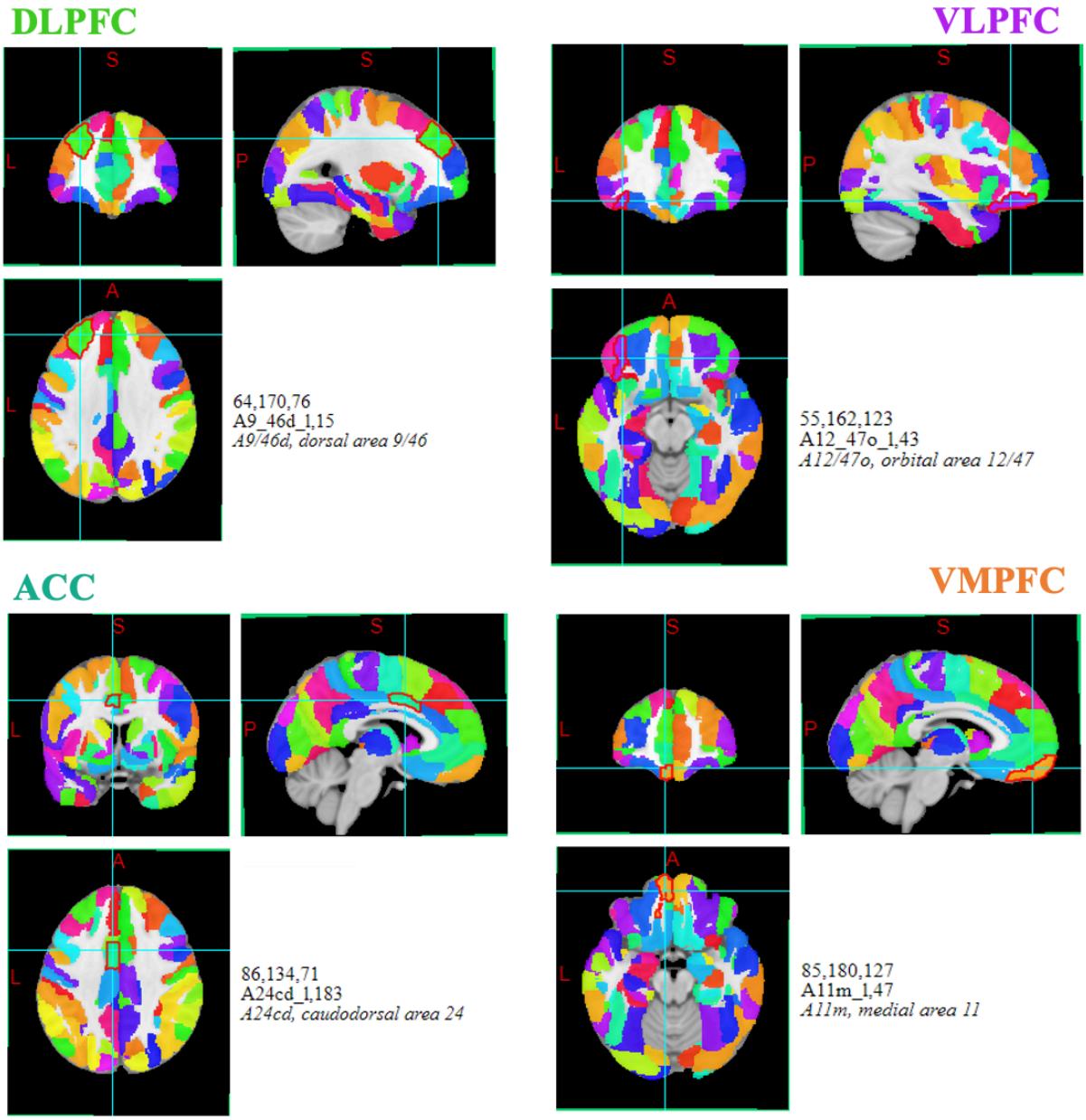

Figure 5. Brainnetome ROI annatomical location for each ROI used in analyses.

The CDR values for each of the six ROIs (left and right were averaged together for VMPFC and dorsal ACC) were then analyzed using a multilevel regression model conducted in R using *lme4* (Bates, Mächler, Bolker, & Walker, 2015), with a random intercept at the participant level to account for the repeated measures design. CDR values during the target condition were included in the model as the DV. To control for non-self-regulatory activity, the

control condition CDR values were entered as a covariate in the model so that only unique variance associated with the target condition was analyzed. The CDR values of both the target and control conditions were log-transformed because the values were positively skewed. An additional covariate was entered into the model to control for differences related to accuracies during the target conditions. Two within-subjects IVs were entered in the model: self-regulation strategy (proactive control, reactive control, inhibitory control, and response conflict) and ROI (dorsal ACC, VMPFC, right hemisphere DLPFC, left hemisphere DLPFC, right hemisphere VLPFC, and left hemisphere VLPFC). Model comparison was conducted to compare each subsequent model as variables were added and assessed against the previous simpler model with the chi-square likelihood ratio test. Each variable created significantly better fit to the data except for gender and trial count, which were then removed for final analyses. The final optimal model contained a random intercept at the participant level, two fixed-effects covariates: control condition CDR values (log-transformed), target condition accuracy, two fixed-effects IVs: self-regulation strategy (four levels) and ROI (six levels), and one interaction term between self-regulation strategy and ROI.

The alpha-level for all analyses was set at 0.05. Since there was a significant interaction between the two factors, post-hoc analyses were performed using the *emmeans* package in R (Lenth, 2020) with a Bonferroni correction for multiple comparisons. Finally, the model assumptions for the multilevel regression model such as homogeneity of variance, linearity and normality of residuals were verified and there were no extreme outliers identified using cook's distance (after removal of one outlier subject described in the Methods above).

CHAPTER 3

RESULTS

Behavior

Accuracy. A 3 (PC, IC, RespC) x 2 (target, control) repeated-measures ANOVA was run comparing condition with self-regulation strategy for accuracy using a linear mixed model with a random-intercept for participants. There was a significant main effect of condition, $F(1, 655) = 1167.48$, $p < .0001$, a significant main effect of self-regulation, $F(1, 655) = 273.64$, $p < .0001$, and a significant interaction between them, $F(2, 655) = 420.11$, $p < .0001$. The same ANOVA was run with PC replaced by RC, and there was a significant main effect of condition, $F(1, 655) = 2231.64$, $p < .0001$, a significant main effect of self-regulation, $F(2, 655) = 58.54$, $p < .0001$, and a significant interaction between them, $F(2, 655) = 230.34$, $p < .0001$. Paired samples *t*-tests revealed significantly lower accuracy in the target condition compared to the control condition across all four self-regulation strategies (all $p < .0001$), suggesting we administered the tasks correctly. When comparing accuracy for the self-regulation strategies in the target condition, PC had the highest accuracy, which was significantly higher than RespC, IC, and RC. RespC was significantly higher than IC and RC. IC was significantly higher compared to RC. When comparing accuracy for the strategies in the control condition, IC had the highest accuracy, which was significantly higher than PC/RC and RespC. RespC was significantly lower in accuracy in the control condition compared to PC/RC. All *t*-test comparisons for accuracy can be found in Table 2. To confirm that there were no gender differences in accuracy, a *t*-test was performed comparing accuracy between males and females, $t(1038) = -0.11$, $p = .91$, therefore it was not included in further analyses as a covariate.

Table 2

Pairwise comparisons (t-test values) for accuracy for each condition

Target

Variable	*M*	*SD*	1	2	3	4
1. IC	59.82	10.67	-			
2. PC	90.57	8.29	22.69****	-		
3. RC	56.02	13.95	-2.73**	23.0****	-	
4. RespC	78.03	5.77	18.16****	14.27****	17.07****	-

Control

Variable	*M*	*SD*	1	2	3	4
1. IC	96.98	5.86	-			
2. PC^	94.33	4.6	5.64****	-		
3. RC^	94.33	4.6	5.64****	NA	-	
4. RespC	88.24	5.95	13.83****	10.08****	10.08****	-

Note. ^PC and RC had the same control condition. All degrees of freedom were equal to 131. All significance was corrected for multiple comparisons using Bonferroni method. Asterisks indicate significance $p < .05$ (*), $p < .01$ (**), $p < .001$ (***) $p < .0001$ (****).

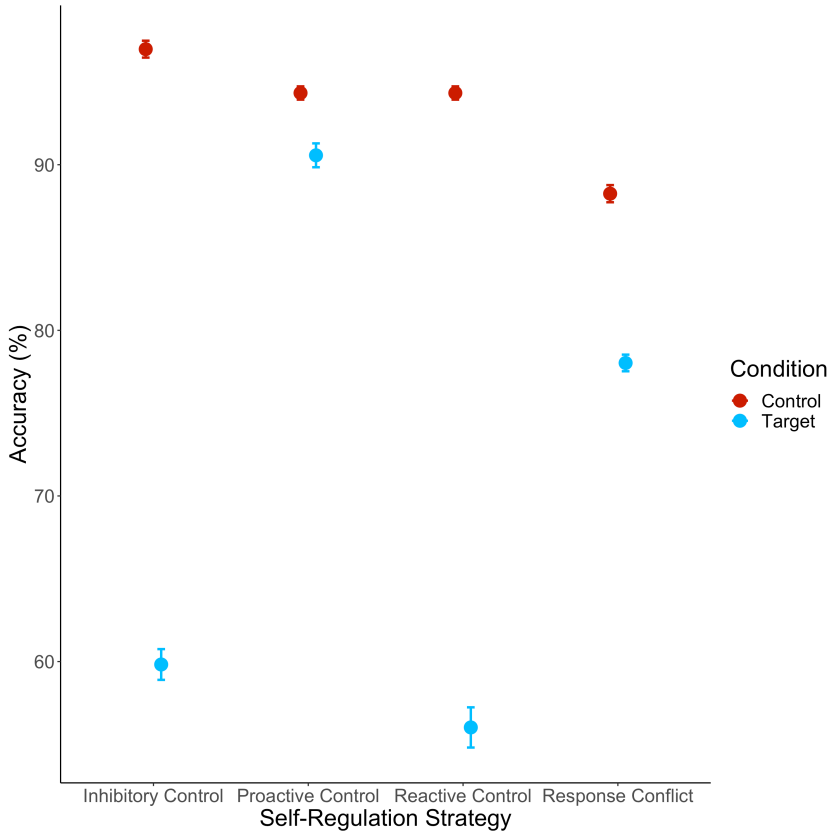

Figure 6. Mean accuracies (% correct). Error bars represent SE.

Reaction Times. A 2 (target vs control) x 2 (PC vs. RespC) repeated measures ANOVA was run comparing condition with self-regulation strategy for RTs using a linear mixed model with a random-intercept for participants. All RTs were calculated from the correct trials only. A significant main effect of condition was found, $F(1, 393) = 5.48$, p=.02, a significant main effect of self-regulation was found, $F(1, 393) = 1233.71$, $p < .0001$, and a significant interaction was found, $F(1, 393) = 59.80$ $p < .0001$. Additionally, two (target vs control) by two (RC vs. RespC) repeated measures ANOVA was run comparing condition with self-regulation for RTs. A significant main effect of condition was found, $F(1, 393) = 179.47$, $p < .0001$, a significant main effect of self-regulation was found, $F(1, 393) = 475.81$, $p < .0001$, and a significant interaction was found, $F(1, 393) = 57.84$ $p < .0001$. Paired samples t-tests revealed significant increased reaction times (RT) for the RC target condition compared to the control condition. The same significant condition difference was found for the RespC target condition vs. the control condition. For PC, the target condition was a significantly faster RT compared to the control condition. A repeated-measures ANOVA was run for one factor (self-regulation strategy) across the target condition revealing a significant effect of self-regulation, $F(1,131) = 1362.5$, $p <.0001$. Subsequent paired samples t-tests comparing RTs in each self-regulation strategy in the target condition revealed significant differences between all three, with RespC having the slowest RT and PC having the fastest RT. In the control condition, since PC and RC used the same control condition (AX), one t-test was performed showing RespC was significantly slower. All *t*-test comparisons for reaction times can be found in Table 3. To confirm that there were no gender differences in RT, a t-test was performed between males and females for RT, $t(892.34) = -1.68$, $p = .09$, therefore it was not included in further analyses as a covariate.

Table 3

Pairwise comparisons (t-test values) for reaction time between target and control for each strategy

Variable	*M*	*SD*	1	2	3	4	5	6
1. Target PC	244.96	60.51	-					
2. Control PC^	448.71	48	23.22****	-				
3. Target RC	539.67	102.8	36.91****	NA	-			
4. Control RC^	314.24	54.19	NA	NA	-24.07****	-		
5. Target RespC	448.71	48	23.81****	NA	-8.89****	NA	-	
6. Control RespC	502.59	89.84	NA	-17.11****	NA	-17.11****	-12.16****	-

Note. ^PC and RC had the same control condition. All degrees of freedom were equal to 131. All significance was corrected for multiple comparisons using the Bonferroni method. Asterisks indicate significance $p < .05$ (*), $p < .01$ (**), $p < .001$ (***) $p < .0001$ (****).

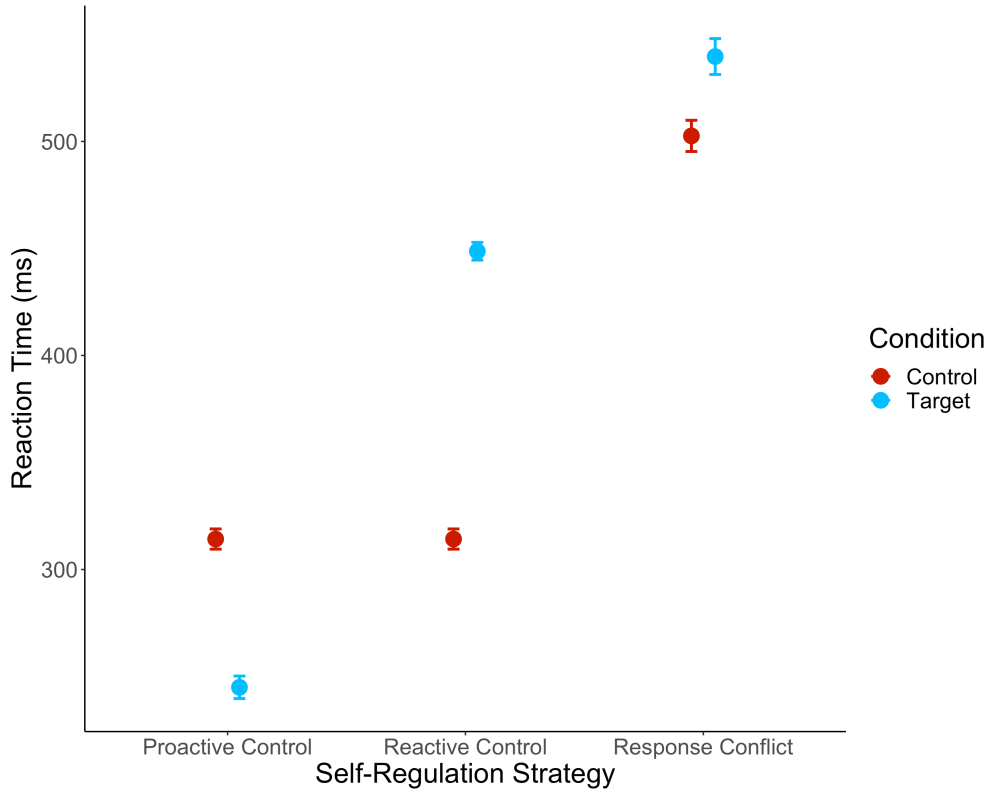

Figure 7. Mean reaction times during correct trials. Error bars represent SE.

N2

A significant main effect of self-regulation strategy on N2 mean amplitude in the target condition was found in the one factor (four levels: IC, PC, RC, RespC) repeated-measures ANCOVA after controlling for N2 mean amplitude in the control condition and accuracy in the target condition, $F(3, 439.45) = 33.15$, $p < .0001$ (Figure 8). PC had the largest (most negative)

N2 amplitude, which was significantly larger than RC and RespC. IC had the smallest (least negative) N2 amplitude. There was no significant difference between RC and RespC mean N2 amplitude. All pairwise comparisons for N2 are reported in Table 4. To confirm that there were no gender differences in target N2 mean amplitude, a *t*-test was performed between males and females for target N2 mean amplitude, $t(521.26) = 1.29$, $p = .20$. Additionally, as low trial counts can contribute to ERP amplitude, Pearson correlation tests were run for each self-regulation strategy to check for relationships between target N2 amplitudes and target trial counts. No significant correlations were found (all $p>.05$); therefore, trial count was not included as a covariate in any analyses.

Table 4

Pairwise t-test comparisons (df) of N2 for each strategy

Variable	1	2	3	4
1. IC	-			
2. PC	9.06**** (509)	-		
3. RC	6.27**** (402)	-4.26*** (518)	-	
4. RespC	6.81**** (463)	-4.55**** (431)	1.26 (487)	-

Note. All significance was corrected for multiple comparisons using Bonferroni method. Asterisks indicate significance $p < .05$ (*), $p < .01$ (**), $p < .001$ (***) $p < .0001$ (****).

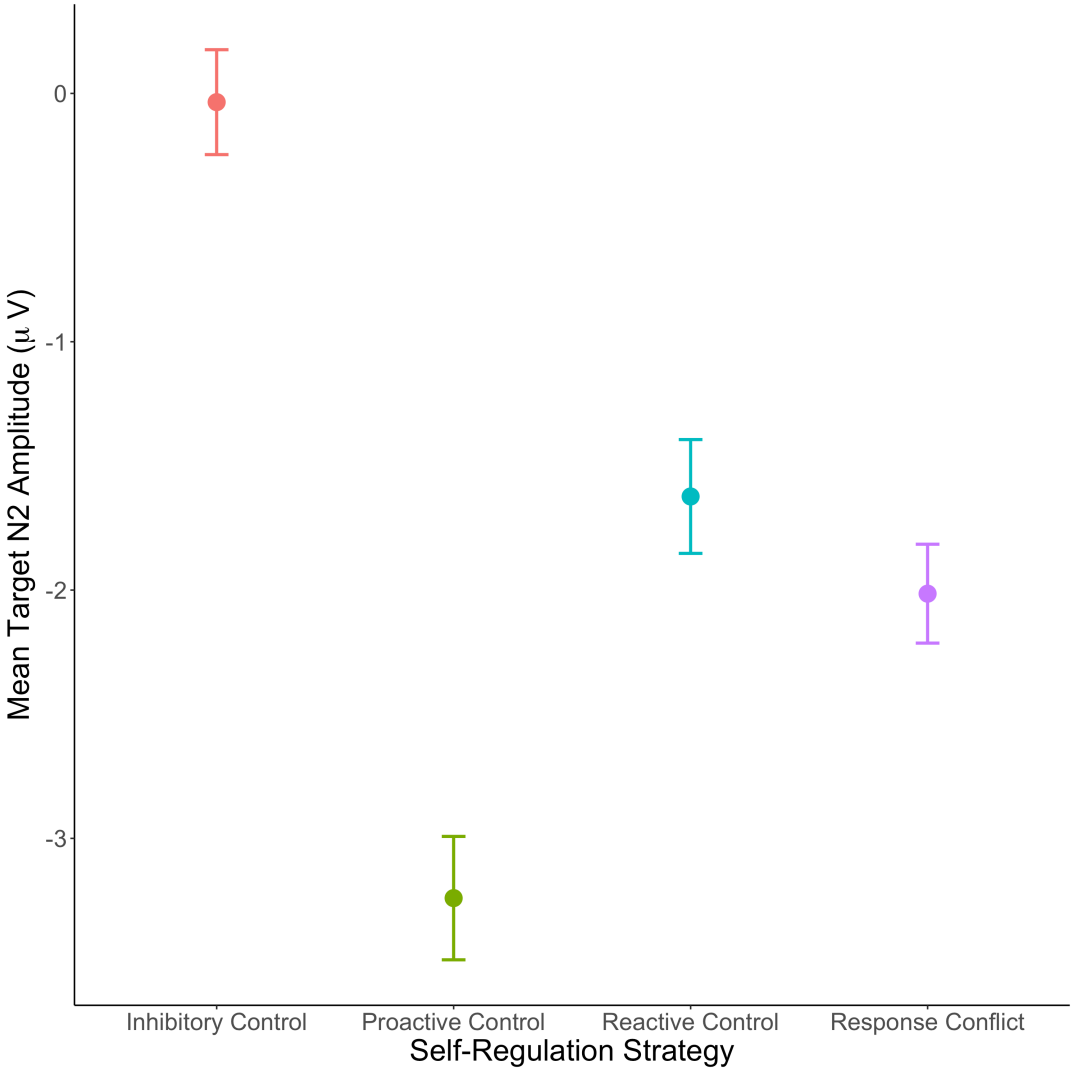

Figure 8. Main effect of self-regulation strategy on mean N2 amplitude. Error bars represent SE. Results are reported with the inclusion of target accuracy and control N2 mean amplitude as covariates.

Figure 9. Mean amplitude for the N2 component for each condition. Error bars represent SE. This is for visualization of the condition differences only; the control condition was entered as a covariate in the model comparing self-regulation strategies.

Source Localization

A base multilevel regression model (target condition CDR values predicted by a random-intercept) was first run to compare to other models to assess model fit. The base model had an interclass coefficient (ICC) of 0.17, anything above 0.1 is sufficient to justify modeling a random intercept. I proceeded to add the covariates and each predictor to the model sequentially, and each subsequent model provided significantly better fit using the chi-square goodness of fit test. Gender was left out of the final model because it did not provide significantly better fit when added during model comparison. Trial counts were left out of the final model as a covariate

because there was no significant relationship between trial count and N2 mean amplitudes. The final model including the random-intercept, two covariates (control condition CDR values and target accuracy), and two within-subjects IVs (self-regulation [SR] strategy and ROI) had a pseudo-R^2 value of 0.66, indicating that the final model explained 66% more of the variance compared to the base model. The full multi-level model effects table and model comparisons table can be found in Appendix B.

The results of the multilevel regression model (after controlling for control condition CDR values and target accuracy) revealed two significant main effects, for self-regulation $F(3,3079.1) = 16.88$, $p < .0001$ (Figure 10), for ROI $F(5, 2957) = 23.47$, $p < .0001$ (Figure 11), and a significant interaction between self-regulation strategy and ROI for the target condition CDR values, $F(15, 3029.9) = 11.18$, $p < .0001$ (Table 5). Follow-up pairwise comparisons between estimated marginal means (Table 6) were conducted for each self-regulation strategy at each level of ROI (Figure 12), and for each ROI at each level of self-regulation (Figure 13). All pairwise comparisons are reported in Table 9 and Table 10. To summarize, significant differences across the self-regulation strategies were found in all ROIs except in VMPFC. For the dorsal ACC, PC and RespC had the greatest activation (CDR), higher than RC and IC. In both the left and right hemispheres of DLPFC, PC and RC had greater activation compared to IC and RespC. In the left VLPFC, the only difference was that RC had greater activation than RespC. In the right VLPFC, RespC, RC, and IC all had greater activation than PC. Looking within each strategy across the ROIs, PC was defined by greater activation in dorsal ACC compared to all other ROIs. IC, RC, and RespC all had greater activation in right VLPFC, however, RespC also had high activation in dorsal ACC.

Table 5

Type III analysis of variance table

Variables	*MS*	*df1*	*df2*	*F*
Control CDR	247.34	1	1988.3	1325.14****
Target Accuracy	2.07	1	2158	11.08****
SR	3.15	3	3079.1	16.88****
ROI	4.38	5	2957	23.47****
SR×ROI	2.09	15	3029.9	11.18****

Note. This ANCOVA was run with the Satterthwaite's method. The dependent variable entered was the target condition CDR values (log-transformed). The first two variables were entered as covariates. The significant main effects of the two factors were qualified by the significant interaction between self-regulation strategy and ROI, $p < .0001$ (****).

Table 6

Estimated marginal means for target CDR values for each SR strategy and ROI

			95% CI	
IC	*M*	*SE*	*LL*	*UL*
ACC	37.47	1.57	34.51	40.68
L DLPFC	37.64	1.74	34.38	41.20
R DLPFC	36.66	1.65	33.55	40.05
L VLPFC	47.34	1.99	43.59	51.41
R VLPFC	65.12	2.75	59.94	70.76
VMPFC	47.22	1.95	43.54	51.21
PC				
ACC	60.31	2.64	55.36	65.71
L DLPFC	44.90	2.14	40.90	49.29
R DLPFC	47.61	2.23	43.42	52.20
L VLPFC	49.46	2.16	45.39	53.89
R VLPFC	50.32	2.24	46.11	54.92
VMPFC	46.94	2.03	43.12	51.10
RC				
ACC	44.56	1.89	41.00	48.44
L DLPFC	44.67	2.06	40.80	48.90
R DLPFC	44.57	2.02	40.77	48.72
L VLPFC	53.52	2.27	49.25	58.16
R VLPFC	65.24	2.90	59.79	71.18
VMPFC	54.31	2.30	49.99	59.00
RespC				
ACC	52.25	2.14	48.21	56.63
L DLPFC	34.66	1.56	31.73	37.86
R DLPFC	36.72	1.61	33.69	40.02
L VLPFC	43.90	1.81	40.48	47.60
R VLPFC	62.43	2.59	57.55	67.72
VMPFC	51.05	2.08	47.13	55.29

Table 7

Pairwise t-test comparisons (df) of Target CDR for each strategy

Variable	1	2	3	4
1. IC	-			
2. PC	-3.53** (2963)	-		
3. RC	6.05**** (3095)	-0.52 (2887)	-	
4. RespC	-1.35 (3183)	3.35** (3184)	3.52** (3137)	-

Note. All significance was corrected for multiple comparisons using Bonferroni method. Asterisks indicate significance $p < .05$ (*), $p < .01$ (**), $p < .001$ (***) $p < .0001$ (****).

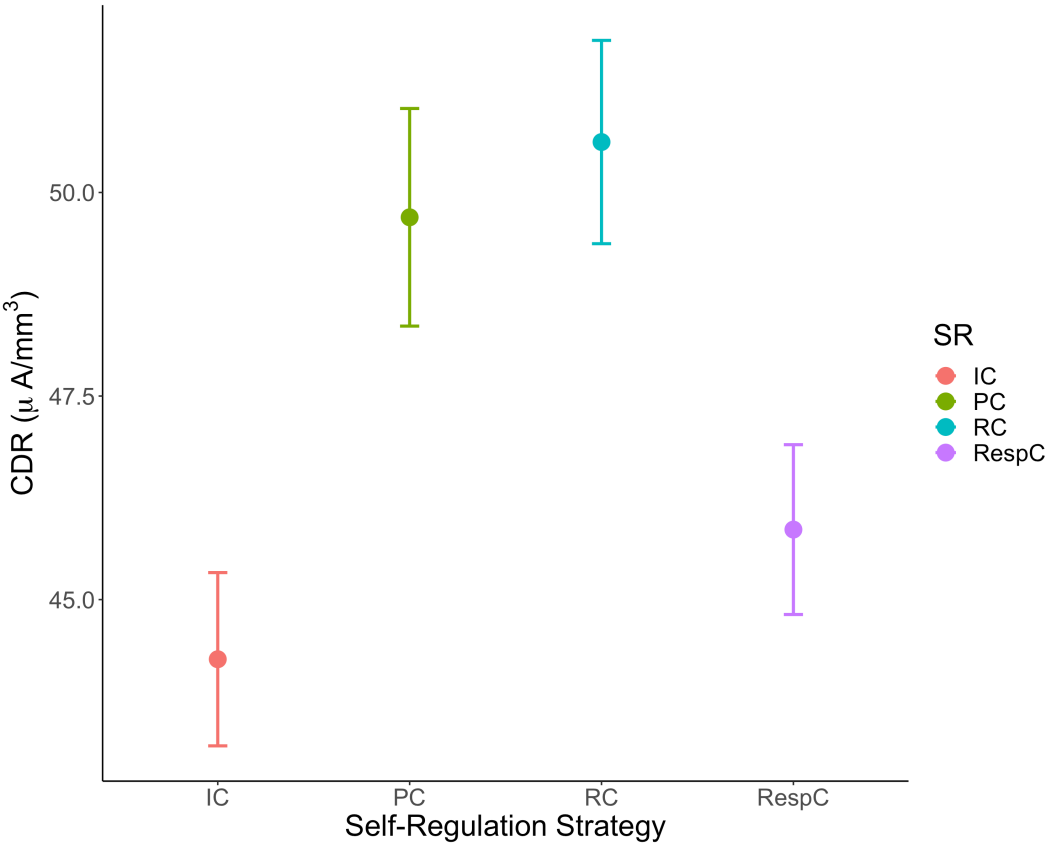

Figure 10. Main effect of self-regulation strategy on CDR. Error bars represent SE.

Table 8

Pairwise t-test comparisons (df) of Target CDR for each ROI

Variable	1	2	3	4	5	6
1. ACC	-					
2. L_DLPFC	5.84**** (3194)	-				
3. R_DLPFC	5.27**** (3181)	-0.82 (3068)	-			
4. L_VLPFC	-0.42 (3061)	-6.25**** (3192)	-5.68**** (3178)	-		
5. R_VLPFC	-7.40**** (3184)	-10.12**** (2888)	-9.97**** (2946)	-7.00**** (3180)	-	
6. VMPFC	-1.40 (3129)	-6.32**** (3124)	-5.90**** (3162)	-1.00 (3135)	6.85**** (3160)	-

Note. All significance was corrected for multiple comparisons using Bonferroni method. Asterisks indicate significance $p < .05$ (*), $p < .01$ (**), $p < .001$ (***) $p < .0001$ (****).

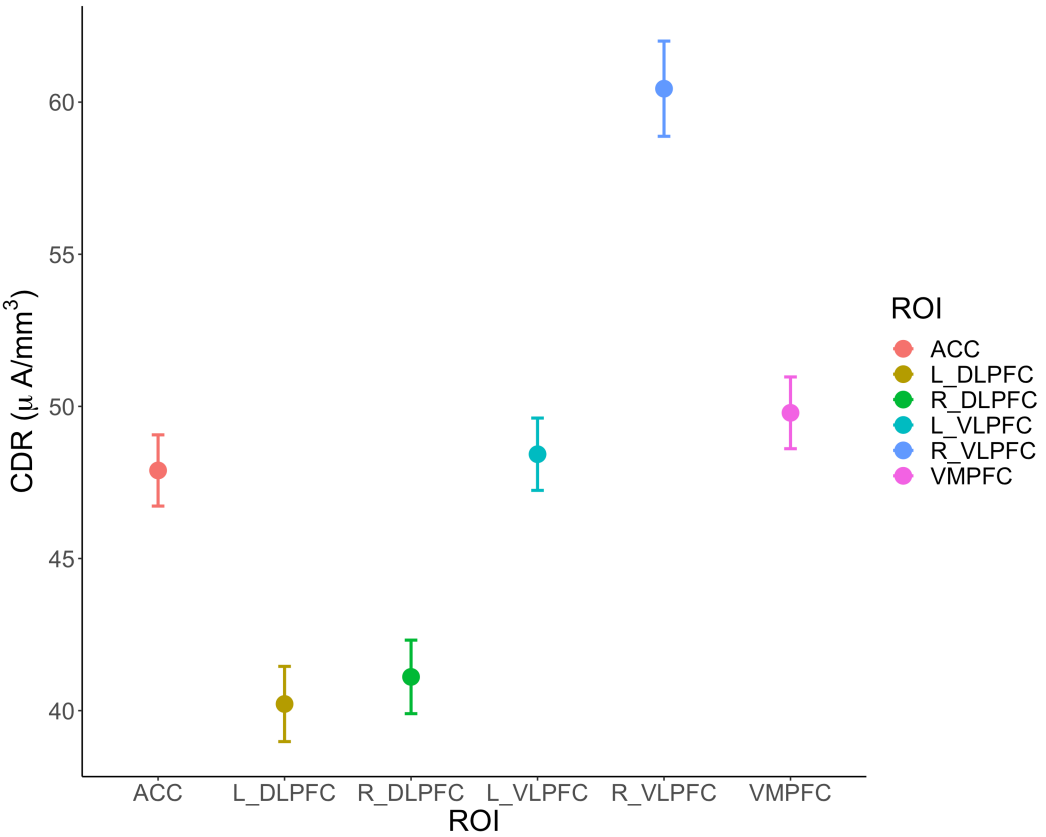

Figure 11. Main effect of ROI on CDR. Error bars represent SE.

Table 9

Pairwise t-test comparisons (df) for each strategy within each ROI

ACC				
Variable	1	2	3	4
1. IC	-			
2. PC	-8.10**** (3184)	-		
3. RC	-3.24* (3065)	5.04**** (3192)	-	
4. RespC	-6.01**** (3127)	2.64 (3095)	-2.83* (3146)	-
L DLPFC				
Variable	1	2	3	4
1. IC	-			
2. PC	-3.00* (3184)	-		
3. RC	-3.20** (3065)	0.09 (3192)	-	
4. RespC	1.49 (3125)	4.77**** (3094)	4.51**** (3146)	-
R DLPFC				
Variable	1	2	3	4
1. IC	-			
2. PC	-4.45*** (3184)	-		
3. RC	-3.66** (3064)	1.10 (3192)	-	
4. RespC	-0.03 (3126)	4.78**** (3095)	3.45** (3146)	-
L VLPFC				
Variable	1	2	3	4
1. IC	-			
2. PC	-0.74 (3184)	-		
3. RC	-2.29 (3067)	-1.31 (3192)	-	
4. RespC	1.37 (3125)	2.20 (3094)	3.52** (3146)	-
R VLPFC				
Variable	1	2	3	4
1. IC	-			
2. PC	4.39*** (3184)	-		
3. RC	-0.03 (3075)	-4.32*** (3192)	-	
4. RespC	0.77 (3125)	-3.96** (3098)	0.78 (3152)	-
VMPFC				
Variable	1	2	3	4
1. IC	-			
2. PC	0.10 (3184)	-		
3. RC	-2.61 (3070)	-2.42 (3192)	-	
4. RespC	-1.49 (3125)	-1.54 (3097)	1.10 (3149)	-

Note. All significance was corrected for multiple comparisons using Bonferroni method. The degrees of freedom method used was the Kenward-Roger method. Asterisks indicate significance $p < .05$ (*), $p < .01$ (**), $p < .001$ (***) $p < .0001$ (****).

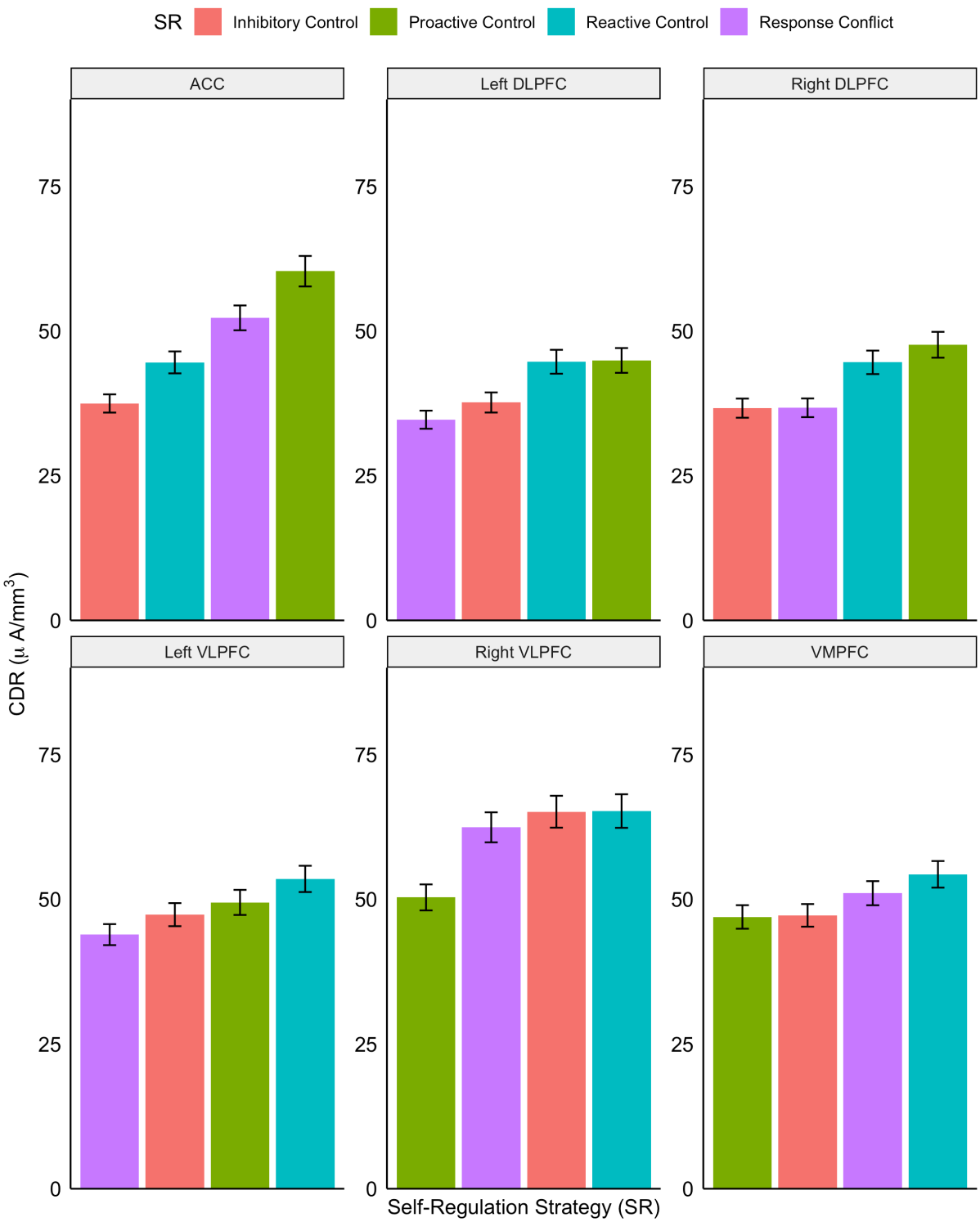

Figure 12. SR patterns of CDR (measure of current) for each ROI**.** Error bars represent SE.

Table 10

Pairwise t-test comparisons (df) for each ROI within each strategy

Inhibitory Control						
Variable	1	2	3	4	5	6
1. ACC	-					
2. L_DLPFC	-0.08 (3120)	-				
3. R_DLPFC	0.40 (3103)	0.50 (3063)	-			
4. L_VLPFC	-4.38*** (3061)	-4.18*** (3116)	-4.70**** (3100)	-		
5. R_VLPFC	-9.91**** (3142)	-9.00**** (3194)	-9.61**** (3193)	-5.70**** (3145)	-	
6. VMPFC	-4.30*** (3079)	-3.98** (3168)	-4.50*** (3153)	0.05 (3082)	5.93**** (3092)	-
Proactive Control						
Variable	1	2	3	4	5	6
1. ACC	-					
2. L_DLPFC	5.36**** (3123)	-				
3. R_DLPFC	4.32*** (3111)	-1.10 (3062)	-			
4. L_VLPFC	3.72** (3061)	-1.76 (3120)	-0.70 (3108)	-		
5. R_VLPFC	3.23* (3150)	-1.85 (3191)	-0.91 (3194)	-0.31 (3152)	-	
6. VMPFC	4.64*** (3085)	-0.77 (3177)	0.25 (3167)	0.97 (3087)	1.28 (3092)	-
Reactive Control						
Variable	1	2	3	4	5	6
1. ACC	-					
2. L_DLPFC	-0.04 (3119)	-				
3. R_DLPFC	-0.01 (3105)	0.04 (3062)	-			
4. L_VLPFC	-3.43** (3061)	-3.27* (3127)	-3.34* (3114)	-		
5. R_VLPFC	-6.68**** (3168)	-6.03**** (3184)	-6.16**** (3190)	-3.50** (3161)	-	
6. VMPFC	-3.65* (3095)	-3.37* (3181)	-3.45** (3172)	-0.27 (3088)	3.37* (3101)	-
Response Conflict						
Variable	1	2	3	4	5	6
1. ACC	-					
2. L_DLPFC	7.43**** (3128)	-				
3. R_DLPFC	6.46**** (3108)	-1.08 (3064)	-			
4. L_VLPFC	3.26* (3062)	-4.31*** (3114)	-3.29* (3094)	-		
5. R_VLPFC	-3.24* (3118)	-9.82**** (3194)	-9.04**** (3188)	-6.35**** (3133)	-	
6. VMPFC	0.44 (3067)	-6.86**** (3157)	-5.92**** (3137)	-2.81 (3075)	3.72** (3090)	-

Note. All significance was corrected for multiple comparisons using Bonferroni method. The degrees of freedom method used was the Kenward-Roger method. Asterisks indicate significance $p < .05$ (*), $p < .01$ (**), $p < .001$ (***) $p < .0001$ (****).

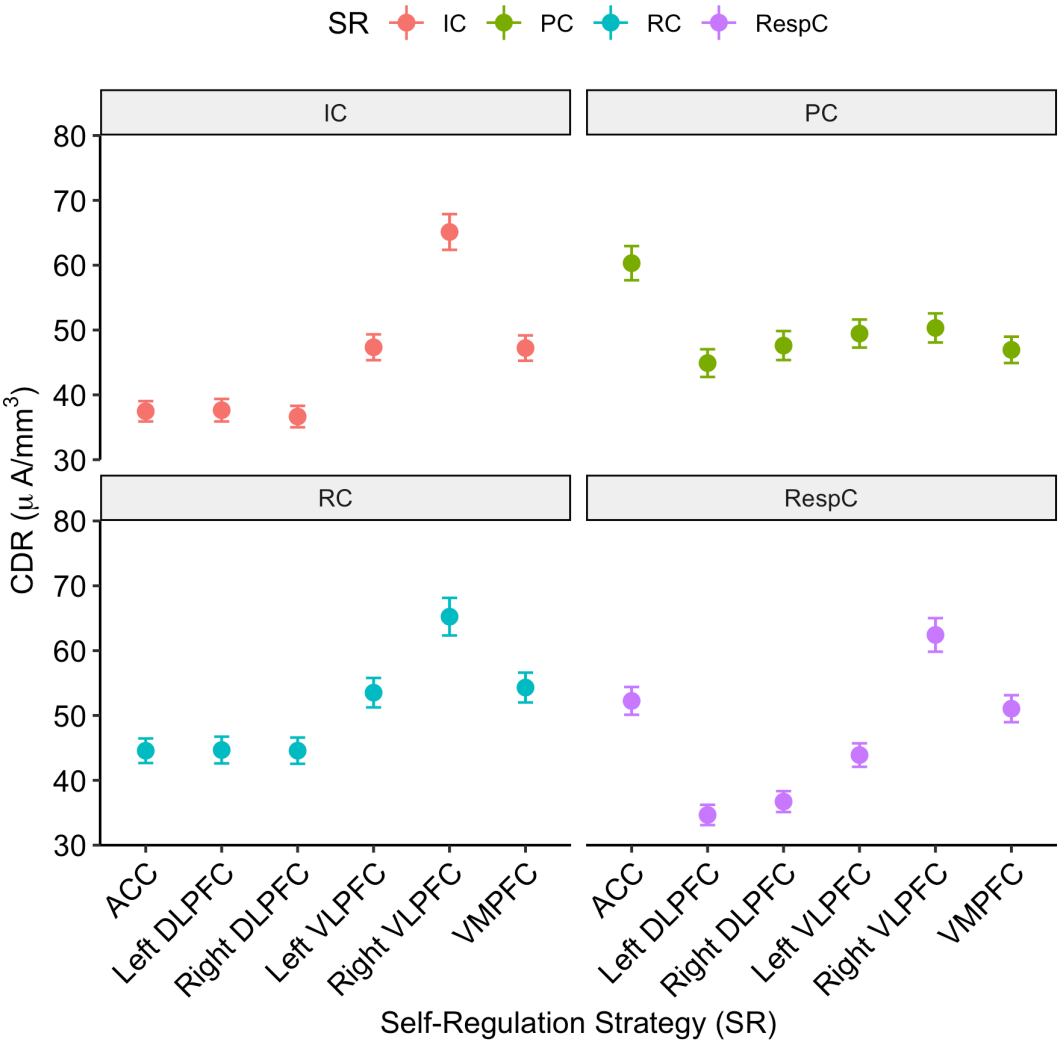

Figure 13. ROI patterns of CDR (measure of current) for each SR strategy**.** Error bars represent SE.

CHAPTER 4

DISCUSSION

The central aim of this exploratory study was to identify variation in ROI engagement in the brain across the contexts of four different self-regulatory strategies. EEG was recorded while participants performed three tasks engaging four self-regulation strategies: proactive and reactive control, inhibitory control, and resolving response conflict. Behavioral data was collected to confirm participant's engagement in self-regulation during each task. The N2 ERP component was extracted for each participant across the tasks as a neural measure associated with self-regulation (Falkenstein, 2006; Lamm, Pine, & Fox, 2013; Nieuwenhuis et al., 2003; Wouwe, Band, & Ridderinkhof, 2009). Ultimately, the neural sources of the N2 were analyzed using source-space analyses to investigate any differences in brain activation during the varying self-regulation tasks. The current study builds on the work by Wagner et al. (2005), using a substantially larger sample size and used ERPs rather than fMRI. Based on the work by Wager and colleagues, I hypothesized differences in prefrontal and anterior cingulate activation during the engagement of the various self-regulation strategies. There was a significant interaction between self-regulation strategy and ROIs in the analyzed model and post-hoc testing revealed different patterns of self-regulation across the ROIs. The ROIs with the largest differences were in the right hemisphere dorsal ACC, VLPFC, and DLPFC, suggesting that these three areas have unique roles in each context of self-regulation.

The most interesting finding of this study was that there were significant differences in dorsal ACC activation across the self-regulation strategies, particularly the strongest activation being in proactive control. The dorsal ACC has been studied in many contexts and its function has been mainly attributed to conflict monitoring (Gehring & Knight, 2000; MacDonald et al.,

2000) and error processing (Carter et al., 1998; Menon et al., 2001). However, Botvinick et al. (2004) emphasized ACC's role across many different forms of conflict, revealing that there is disagreement in the literature about whether ACC is more involved in cognitive function or motor function of control. Previous research also suggests that the ACC is involved in general performance monitoring (MacDonald et al., 2000; Oliveira, McDonald, & Goodman, 2007). I expected to find the dorsal ACC to be most activated during reactive control and resolving response conflict because 1) these strategies showed the lowest accuracy levels, i.e., highest error processing, and 2) they were elicited in the context of direct conflict, i.e., which button to press. Instead, I found the most ACC activation in proactive control and the next highest activation in response conflict. This indicates that there may be more similarities in the contexts of these two self-regulation strategies based on the ACC activation. In the context of response conflict, the ACC appears (as in previous studies) to aid in preventing errors on incongruent trials. This also aligns with the ACC's involvement in top-down control and focused attention (Kerns et al., 2004; Schulz et al., 2011). It is critical during response conflict to be highly attentive to the current stimulus and exert top-down control to respond correctly to the current stimulus. The ACC activity during proactive control is best understood in the context of the AX-CPT task. During the task, most trials are A followed by X. However, during a subset of those trials B appears first and then X. This is where proactive control is engaged, during the appearance of B. This is when the participant must recognize the deviation from the typical stimulus appearance of A, and remember for the subsequent X stimulus that the correct response is different from the typical A followed by X trial. This trial type requires a heightened need for focused attention and prevention of a following error over a delayed period of time, similar to the process of performance monitoring, which requires dorsal ACC activity.

The right VLPFC revealed lower activation during proactive control compared to the rest of the self-regulation strategies but no other differences. The main role of right VLPFC is in conflict adaptation (Egner, 2011) and motor inhibition (Aron, Robbins & Poldrack, 2004). The difference in activation found in the current study indicates that inhibitory control, reactive control, and response conflict, all have similar requirements of the right VLPFC for resolving conflict. Inhibitory control actively involves motor inhibition, and reactive control and response conflict both include more conflict adaptation (as evidenced by lower accuracy). Therefore, these findings indicate these processes require a similar recruitment of right VLPFC activation (similar to Levy & Wagner, 2011), while proactive control is distinctly different.

Although lateralization of DLPFC was examined for this study, because previous studies have found different processes localized to the left vs. the right hemisphere (MacDonald et al. 2000; Vanderhasselt, De Raedt, & Baeken, 2009), the results of this study did not find differences in ROI activation patterns between the left and the right hemispheres. There was significantly greater activation of both left and right DLPFC during proactive and reactive control, and less during inhibitory control and response conflict. This finding aligns with previous studies indicating that DLPFC is important in updating goal information using attentional processes and top-down control (Miller & Cohen, 2001; Sakai & Passingham, 2006; D'Esposito & Postle, 1999). Since proactive and reactive control are both actively engaged to maintain goals, it follows that these two self-regulation strategies would require more DLPFC activation. This brain area has been shown to be important during these strategies in previous studies as well (Braver, Paxton, Locke, & Barch, 2009; Lamm, Pine, & Fox, 2013). In particular, Braver's framework of the dual mechanisms of control outlining the theories behind proactive

and reactive control (Braver, 2012), puts emphasis on the importance of DLPFC's involvement in coordinating between these two types of self-regulation.

The left VLPFC showed one significant difference, with higher activation during reactive control than response conflict, but no differences between the other strategies. This finding is aligned with the findings of Braver and colleagues (2007), who theorized that left VLPFC activity is greater during reactive control engagement. This finding is consistent with the idea that left VLPFC allows for flexible self-regulation to achieve a goal state. The fact that left VLPFC is distinctly different between reactive control and response conflict suggests that left VLPFC is specifically involved in the memory and maintenance of overarching goals (Badre et al., 2005, Badre & Wagner, 2007; D'Esposito et al., 1999) required during the AY trials of AX-CPT, as opposed to those involved in the hybrid Flanker Global/Local task. Response conflict, as it is measured during the hybrid Flanker Global/Local task, is a more immediate resolution of conflict between the cue and the stimulus. The left VLPFC activity captures the difference between response conflict and reactive control, which is more about keeping the memory of the previous cue in mind and resolving the probe conflict, while also maintaining a goal state.

VMPFC did not reveal any significant differences between the strategies, which is in line with the current literature. VMPFC has been shown to be involved mainly in the context of behavioral regulation in an emotional context (Dolan & Park, 2002). Since none of the tasks in this study had any emotional components to them, I suspected that there would be little to no involvement of VMPFC. I did hypothesize that VMPFC may be modulated by differences in DLPFC (Hare, Camerer, and Rangel, 2009); however, we did not find evidence for that in this study.

Results for the scalp-level N2 activation revealed a unique pattern of negative amplitudes, where proactive control elicited the largest (most negative) N2 amplitudes. Typically, larger N2s have been shown to be related to higher conflict conditions (Van Veen & Carter, 2002; Falkenstein, 2006; Lamm, Pine, & Fox, 2013). However, here proactive control appears to have the least conflict (with fast reaction times and high accuracy) compared to the other self-regulation strategies but has the largest N2 amplitude. Reactive control and response conflict had similar N2 amplitudes, which both had more associated conflict (slower reaction times and lower accuracy in the target condition). Another possible explanation is that N2 amplitudes reflect more broadly, higher levels of cognitive control (Folestein & Van Petten, 2008) and that because proactive control engaged more attention and effort related to cognitive control, it also showed larger N2s. Additionally, the N2 is commonly source localized to the ACC (Van Veen & Carter, 2002; Yeung et al., 2004) and as discussed above, the dorsal ACC activation follows the N2 pattern, with the greatest activation elicited during proactive control. Another potential reason that the N2 is greatest for proactive control, could be that as it requires you to actively maintain goal information, it requires sustained brain activation. I did not examine timing in this study; however, Lamm et al. (2013) observed increased DLPFC activation during the delay period following the cue, more so than the cue itself, using an AX-CPT. In other studies, using fMRI (such as Braver, 2009), this timing difference is blurred because of fMRI's low temporal resolution and averaging of brain activation across longer time periods.

The behavioral results of the study indicated that self-regulation was engaged. Accuracy was lower in the target trials, trials where self-regulation was required compared to control trials. However, the pattern of differences between target and control was different across the self-

regulation strategies, specifically, proactive control had the smallest difference, while inhibitory and reactive control had large differences. This is why accuracy on target trials was included as a covariate in the final model assessing differences in ROI activation. This allowed for the examining of differences in ROIs reported across self-regulation strategy beyond any differences in accuracy. The reaction time results revealed consistent patterns in reactive control and response conflict, with slower reaction times during the target trial, indicating that self-regulation was engaged. Proactive control showed the opposite effect, where the target condition showed faster reaction times. However, this result is to be expected in the context of AX-CPT (similar effect found in Cudo et al. (2018) and Lamm et al. (2013)). During the target trials of proactive control, participants know what button to press after the following probe because of the cue that was given, which gives them time to plan their response leading to a quicker reaction time. However, during the control trial once the cue is presented participants must wait until they see the following probe to understand what button to press, leading to a slower reaction time.

Overall, the purpose of this study was to better understand contextual changes in four self-regulation strategies based on the differences in ROI activation in the cortex. There are many nuances and differences in neural processes during conflict, cognitive control, and self-regulation (see reviews: Egner, 2008; Heatherton, 2011; Gratton et al., 2017; Ridderinkhof et al., 2004; Botvinick & Cohen, 2014). Berkman, Falk and Lieberman (2012) attempted to analyze three processes similar to the ones examined in the current study, those involved in goal maintenance, performance monitoring, and response inhibition. The authors described goal maintenance as a process that uses cognitive representation and working memory to act according to a goal, whereas performance monitoring is used to monitor the goal state and make up any discrepancies between the current and desired end goal state. Therefore, based on

previous studies it is theorized that the DLPFC and ACC work together to execute these processes. The authors go on to also describe response inhibition and VLPFC's involvement as an area critical to inhibiting an action. The authors in that paper designed one task to engage all three of these processes together instead of how the current study kept them separate. They found a mix of activations across the main ROI areas that was hard to interpret and separate between the three processes. I believe the current study allowed for a more conclusive understanding of the involvement of each area by examining the tasks within subjects. Also, by looking at specific self-regulation strategies, the current study was able to identify differences in brain areas that indicate the relative importance of goal maintenance, performance monitoring, and response inhibition in each of the tasks. In particular, right VLPFC seemed to be critically involved in separating proactive control from the rest of the self-regulation strategies. Based on the right VLPFC activity, inhibitory control, reactive control, and response conflict seem to engage the most conflict adaptation. Similar to previous findings, the DLPFC was important in goal maintenance required during proactive and reactive control, but not as much in response conflict or inhibitory control. Finally, the dorsal ACC differed across all strategies, with the most activity required by proactive control for top-down control and performance monitoring, and the least activity in inhibitory control. It was expected that all three of these areas would be important to self-regulation, but this study in particular revealed the key differences between those regions during different self-regulation strategies.

Limitations and Future Directions

Limitations

Although the source space analysis for this study was conducted with current best practices, there were limitations to the precision of that analysis. First, the best way to reduce noise in source space analysis is to have individual MRI scans. However, I did not have access to an MRI scanner, so instead had to use a template MRI. Additionally, I did not use age-matched MRI templates, so there could be noise induced due to developmental differences in age. However, most of the participants in the sample were between 18 and 21 years, which should only have small differences in development compared to our standardized template MRI, which was age-averaged to about 23 years. Another technique used by many doing source space analyses is to individually map electrode locations in 3D space. However, I did not have access to this information for my participant pool, so again, I had to use a template for a 128-electrode system. Proportionally, all electrodes should fall within the same general area across subjects but there is a slight lack of precision because of this. Another limitation was that about half of the total collected participant data was able to be used in this current study. There were a lot of factors that reduced the final participant number from 287 to 132, including but not limited to: technology failures, incomplete EEG recordings, incompatible hair styles with the EEG equipment, incomplete survey data, large amounts of artifacts during preprocessing of EEG data and low trial accuracies. Finally, due to the limited diversity in the available university participant pool in addition to the restrictions of participation due to particular hair styles' incompatible with EEG collection discussed in the Participants section, there is a bias against minority groups in our sample. Therefore, the conclusions in this study cannot be generalized broadly to a larger and more diverse population. Some research suggests that there are

differences in the development of self-regulation as a function of cultural context (Jaramillo et al., 2017). Future research should place an emphasis on recruiting a more diverse sample to test for any differences in self-regulation strategies across cultures as well as any differences in associated neural activations.

Future Directions

Although this study provided a careful analysis of differences in ROIs for different self-regulation strategies, there is plenty more that could further differentiate the importance of these brain areas across self-regulation. First, the current study could further try to understand the differences in the ROIs across self-regulation strategies by looking at them in the context of behavioral differences. Using correlations between CDR activation and behavioral measures like accuracy and reaction time could provide further insights to the behavioral results of these different neural activations. Additionally, a lot of survey data was collected from the participants of this study, so future research could analyze how these differences in ROIs and self-regulation strategies potentially reflect differences in different types of people. Specifically, a future investigation into the differences in impulsivity as it relates to the differences in ROIs and self-regulation, would be an important study, which could contribute to our understanding of the neural mechanisms of self-regulation and how they may differ in those higher in impulsivity. Finally, similar to Braver and colleagues (2009) study between different age groups, future research into the differences in self-regulation and neural activations in older vs. younger adults would further our understanding of the developmental scope in these differential brain areas and examine any changes in self-regulation related to healthy aging.

Conclusion

The results of the current study revealed important differences between dorsal ACC, DLPFC, and VLPFC activation and how patterns of activation across these regions uniquely contribute to reactive control, proactive control, inhibitory control, and resolving response conflict. Importantly, this study also revealed similarities and differences between the self-regulation strategies based on the differing involvement of ROIs, suggesting that the current practice in the extant literature of "averaging over" self-regulation strategies is problematic and likely causes inconsistencies in the literature.

The overall broader implication of this research was to better understand the neural mechanisms engaged during different contexts of self-regulation and to identify where the brain activation may be similar or different. Understanding these mechanisms help elucidate self-regulation research more broadly in the contexts of self-regulation dysfunction resulting in atypical behaviors seen in those with mental health disorders. Most importantly, these findings shed light on how different prefrontal brain areas are involved in certain strategies. Further investigation using advanced analysis technique (such as latent profile analysis) should be applied to investigate if these differences in brain region activation can be divided into groups within the sample, such as those with differences in impulsivity. Since many mental health disorders involve dysregulation of the behavior, it would be interesting to identify if differences in prefrontal brain activation patterns are associated with differences in self-regulatory behavior.

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APPENDIX

Appendix A: Institutional Review Board approval

UNIVERSITY OF ARKANSAS

Office of Research Compliance
Institutional Review Board

January 30, 2017

MEMORANDUM

TO:	Connie Lamm
FROM:	Ro Windwalker IRB Coordinator
RE:	New Protocol Approval
IRB Protocol #:	17-01-386
Protocol Title:	*Differential Neural Correlates Underlying Various Self-Regulation Strategies*
Review Type:	☐ EXEMPT ☒ EXPEDITED ☐ FULL IRB
Approved Project Period:	Start Date: 01/25/2017 Expiration Date: 01/24/2018

Your protocol has been approved by the IRB. Protocols are approved for a maximum period of one year. If you wish to continue the project past the approved project period (see above), you must submit a request, using the form *Continuing Review for IRB Approved Projects*, prior to the expiration date. This form is available from the IRB Coordinator or on the Research Compliance website (https://vpred.uark.edu/units/rscp/index.php). As a courtesy, you will be sent a reminder two months in advance of that date. However, failure to receive a reminder does not negate your obligation to make the request in sufficient time for review and approval. Federal regulations prohibit retroactive approval of continuation. Failure to receive approval to continue the project prior to the expiration date will result in Termination of the protocol approval. The IRB Coordinator can give you guidance on submission times.

This protocol has been approved for 120 participants. If you wish to make *any* modifications in the approved protocol, including enrolling more than this number, you must seek approval *prior to* implementing those changes. All modifications should be requested in writing (email is acceptable) and must provide sufficient detail to assess the impact of the change.

If you have questions or need any assistance from the IRB, please contact me at 109 MLKG Building, 5-2208, or irb@uark.edu.

109 MLKG • 1 University of Arkansas • Fayetteville, AR 72701-1201 • (479) 575-2208 • Fax (479) 575-6527 • Email irb@uark.edu

UNIVERSITY OF ARKANSAS

To:	Connie Lamm BELL 4188
From:	Douglas James Adams, Chair IRB Committee
Date:	01/27/2020
Action:	**Expedited Approval**
Action Date:	01/23/2020
Protocol #:	1708026820R003
Study Title:	Differential Neural Correlates Underlying Various Self-Regulation Strategies
Expiration Date:	01/24/2021
Last Approval Date:	01/25/2020

The above-referenced protocol has been approved following expedited review by the IRB Committee that oversees research with human subjects.

If the research involves collaboration with another institution then the research cannot commence until the Committee receives written notification of approval from the collaborating institution's IRB.

It is the Principal Investigator's responsibility to obtain review and continued approval before the expiration date.

Protocols are approved for a maximum period of one year. You may not continue any research activity beyond the expiration date without Committee approval. Please submit continuation requests early enough to allow sufficient time for review. Failure to receive approval for continuation before the expiration date will result in the automatic suspension of the approval of this protocol. Information collected following suspension is unapproved research and cannot be reported or published as research data. If you do not wish continued approval, please notify the Committee of the study closure.

Adverse Events: Any serious or unexpected adverse event must be reported to the IRB Committee within 48 hours. All other adverse events should be reported within 10 working days.

Amendments: If you wish to change any aspect of this study, such as the procedures, the consent forms, study personnel, or number of participants, please submit an amendment to the IRB. All changes must be approved by the IRB Committee before they can be initiated.

You must maintain a research file for at least 3 years after completion of the study. This file should include all correspondence with the IRB Committee, original signed consent forms, and study data.

cc: Michelle Gray, Key Personnel
Stephanie M Long, Key Personnel
Morgan Middlebrooks, Key Personnel
Arooj Abid, Key Personnel
Eric Logan Rawls, Key Personnel
Carroll Gene Bentley, Key Personnel
Andrew Wilton Jennings, Key Personnel
Ebony A Walker, Key Personnel
Emily Brianne Tolar, Key Personnel
Erik Lyle Abramson, Key Personnel
Jahnavi Kodali, Key Personnel
Gabriel Keifer Bernardo, Key Personnel

Appendix B: Supplementary Tables

Table 1
Full multi-level model parameter estimates

	log(CDR_Target)		
Predictors	*Estimates*	*CI*	*df*
(Intercept)	1.47 ***	1.31 – 1.64	3140.00
Accuracy_Target	-0.00 ***	-0.00 – -0.00	3140.00
log(CDR_Control)	0.58 ***	0.55 – 0.61	3140.00
IC	*Reference*		
PC	0.48 ***	0.36 – 0.59	3140.00
RC	0.17 **	0.07 – 0.28	3140.00
RespC	0.33 ***	0.22 – 0.44	3140.00
ACC	*Reference*		
L_DLPFC	0.00	-0.10 – 0.11	3140.00
R_DLPFC	-0.02	-0.13 – 0.08	3140.00
L_VLPFC	0.23 ***	0.13 – 0.34	3140.00
R_VLPFC	0.55 ***	0.44 – 0.66	3140.00
VMPFC	0.23 ***	0.13 – 0.34	3140.00
SRPC:ROIL_DLPFC	-0.30 ***	-0.45 – -0.15	3140.00
SRPC:ROIL_VLPFC	-0.43 ***	-0.58 – -0.28	3140.00
SRPC:ROIR_DLPFC	-0.21 **	-0.36 – -0.07	3140.00
SRPC:ROIR_VLPFC	-0.73 ***	-0.88 – -0.59	3140.00
SRPC:ROIVMPFC	-0.48 ***	-0.63 – -0.33	3140.00
SRRC:ROIL_DLPFC	-0.00	-0.15 – 0.15	3140.00
SRRC:ROIL_VLPFC	-0.05	-0.20 – 0.10	3140.00
SRRC:ROIR_DLPFC	0.02	-0.13 – 0.17	3140.00
SRRC:ROIR_VLPFC	-0.17 *	-0.32 – -0.02	3140.00
SRRC:ROIVMPFC	-0.03	-0.18 – 0.11	3140.00
SRRespC:ROIL_DLPFC	-0.42 ***	-0.56 – -0.27	3140.00
SRRespC:ROIL_VLPFC	-0.41 ***	-0.56 – -0.26	3140.00
SRRespC:ROIR_DLPFC	-0.33 ***	-0.48 – -0.18	3140.00
SRRespC:ROIR_VLPFC	-0.37 ***	-0.52 – -0.23	3140.00
SRRespC:ROIVMPFC	-0.25 ***	-0.40 – -0.11	3140.00
Random Effects			
σ^2	0.19		
$\tau_{00\ ID}$	0.03		
ICC	0.12		
N_{ID}	132		
Observations	3168		
Marginal R^2 / Conditional R^2	0.655 / 0.698		

** p<0.05 ** p<0.01 *** p<0.001*

Table 2

Model comparison between nested models

Nested Models	*df*	AIC	BIC	log Likelihood	deviance	Chi-Sq *df*	Chi-Square
Intercept	3	7235.50	7253.70	-3614.80	7229.50	NA	NA
SR	6	7188.20	7224.60	-3588.10	7176.20	3	53.33****
ROI	11	5020.90	5087.60	-2499.50	4998.90	5	2177.27****
SR×ROI	26	4926.50	5084.00	-2437.20	4874.50	15	124.48****
Control CDR	27	3934.00	4097.70	-1940.00	3880.00	1	994.44****
Target Accuracy	28	3925.00	4094.70	-1934.50	3869.00	1	11.06****
Gender	30	3924.50	4106.30	-1932.30	3864.50	2	4.45

Note. Each row indicates the variable added to the model for comparison to the model in the row above. CDR values were log-transformed. Asterisks indicate significance level, $p < .0001$ (****).